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UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF ILLINOIS

UNITED STATES OF AMERICA, Plaintiff, v. NAVISTAR, INC., Defendant.	**Case Number**: 15-CV-6143 **Judge:** Hon. Mary M. Rowland

EXPERT REPORT OF DR. JOEL SCHWARTZ Ph.D.

OCTOBER 2019

Signed: _____

Date: __October 3, 2019_____

INFORMATION REQUIRED BY THE FEDERAL RULES OF CIVIL PROCEDURE

The following is a list of the items required by the Federal Rules of Civil Procedure provided with this report:

(1) This report contains my opinions, conclusions, and the reasons therefore on the subjects that I was asked to address.

(2) The body of the report and Appendices list the data and other information considered in forming these opinions.

(3) Exhibits and tables in summary of, or support of, these opinions are included with this report.

(4) Appendix B provides a full statement of my qualifications.

(5) Appendix B also includes a listing of publications.

(6) I am being compensated for my work on this report and for any testimony at the rate of $210 per hour.

I. Qualifications

I am a Professor in the Departments of Environmental Health and Epidemiology at the Harvard School of Public Health, in the Department of Medicine at Harvard Medical School, Director of the Harvard Center for Risk Analysis, member of the faculty of the Environmental Biostatistics program, and on the Steering Committee of the Harvard University Center for the Environment. I am also a former member of the board of Councilors of the International Society for Environmental Epidemiology, and the Editorial Board of the American Journal of Respiratory and Critical Care Medicine. I have served on three National Academy of Sciences panels. I was a recipient of a John D. and Catherine T. MacArthur Fellowship, and of the John Goldsmith Award for career achievement of the International Society for Environmental Epidemiology. I am the most cited author in the field of air pollution research and a Thompson-Reuters Highly-cited Author. I have over 800 peer-reviewed papers published or in press, which have been cited over 60,000 times in other peer-reviewed publications. Over 700 of these scientific papers have examined air pollution. I have made air pollution a major focus of my research, which ranges from time series studies of daily effects to cohort studies of long-term effects to genetic and epigenetic studies. It also includes atmospheric modeling of pollution, exposure modeling, risk assessment and benefit-cost analyses. These studies varied from those focused on critical events (e.g., deaths, heart attacks) to studies addressing mechanisms and exposure. I have

testified before Congress twice about air pollution, served as an expert advisor in reviews for the World Health Organization, and testified as an expert in the health effects of air pollution in federal court.

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II. SUMMARY OF CONCLUSIONS

Nitrogen oxides are substances formed by combustion. At high temperatures, such as those present in diesel engines, the nitrogen in the air reacts with the oxygen in the air to form nitrogen oxides. Higher temperatures result in more nitrogen oxide formation and diesel engines generally run at higher combustion temperatures than gasoline fueled engines. The two major compounds are NO and NO2 and are collectively referred to as NOx.

NOx effects human health in several ways. First, NOx is itself an air pollutant, with known effects on human health. It is an irritant gas that penetrates deeply into the lung. Second, NOx in the air reacts with other compounds, including volatile organic compounds and ammonia, to form both particles and ozone, which in turn have known effects on human health. These are deemed secondary pollutants, since they are not directly emitted, but form by chemical reactions in the atmosphere. One specific example is reacting with organic gases emitted from plants or unburned fuel from vehicles and forming organic carbon particles, another involves reacting with ammonia to form ammonium nitrate particles. A third involves reaction with oxygen and organic gases to form ozone. Importantly, there are few pure particles in the atmosphere. Particles in the air collide and adhere to each other. In the process the nitrogen-based particle, which is acidic, can transform existing particles. Hence an ammonium nitrate

particle can mix with, for example, a copper particle generated from brake wear and produce copper nitrate.

Assessments of the health effects of NOx emissions have historically focused on its role in the formation of secondary particles and ozone. However, a large and growing literature has developed in recent years regarding the direct health impacts of NOx exposure. Here I summarize my conclusions on all three.

II.A. SUMMARY OF CONCLUSIONS ON $PM_{2.5}$ HEALTH EFFECTS

There is clear, convincing evidence that particulate air pollution from combustion, such as from coal plant boilers, kills people. Further, the number of people it kills each year in the United States is not small—it is larger than the number of deaths each year from AIDS, breast cancer, and prostate cancer put together. Indeed, a recent report in the Lancet estimated that worldwide, it kills more people than AIDS, Malaria and Tuberculosis. Hence, even a fraction of a percent change in this number would be more deaths than occur in a major plane crash.

In addition to killing people, particles trigger heart attacks and strokes; destabilize people with heart failure, driving them into the hospital; lower cognitive function in children; accelerate cognitive decline in the elderly; and increase the risk of asthma attacks and exacerbate respiratory infections, leading to increased hospital admissions for those conditions.

These findings are not due to failure to exclude other possible explanations. Similar associations are seen when we control for other pollutants and weather, when we control for pre-existing disease, smoking, etc. Furthermore, randomized controlled trials have shown that, for example, putting particle filters in homes (vs. sham filters) reduced blood pressure, coagulability of blood, systemic inflammation, stress hormones, constriction of blood vessels, insulin resistance, highly oxidizing chemicals, and produced more stable electrocardiogram patterns. These are all predictors of the increased deaths, heart attacks, strokes, etc. mentioned above.

Moreover, animal and toxicological studies also show changes in intermediary endpoints (e.g., electrocardiogram patterns, measures of inflammation, blood pressure, and hardening of the arteries (atherosclerosis) that are consistent with the changes seen in the epidemiologic studies.

Other studies have demonstrated down to the lowest measured exposure that these effects do not have a threshold, and that the deaths produced by particles are not just being advanced by a few weeks but can reduce the life expectancy of entire populations by several years.

All of this indicates that any increase in particle concentrations has substantial effects on human health, including increasing the risk of early deaths. Details justifying these conclusions are below in section III.

Overall, there have been thousands of studies of the health effects of particles in the last two decades, and the overwhelming scientific consensus is that particles are associated with early deaths, as well as a range of other adverse events. Here, I discuss the overwhelming scientific consensus supporting this conclusion. I later assess its basis, including studies of short and long-term exposure to particulate air pollution and early deaths, as well as respiratory and cardiovascular effects, such as heart attacks, hospital admissions, etc., and cognitive effects. I include a discussion of effect thresholds and the evidence for continuous effects at low concentrations.

II.A.1. THE SCIENTIFIC CONSENSUS

For two decades, there has been an overwhelming scientific consensus about the health effects of particulate air pollution. It is widely accepted that such particles reduce life expectancy, trigger heart attacks, have a wide range of other adverse effects on health, and that sufficient evidence exists to quantitatively estimate the impacts of reducing air pollution on avoided deaths, etc. Several of the most reputable health organizations have noted the consensus on the health effects of particulate matter. For example, as far back as the 2002 World Health Report, the World Health Organization (WHO) concluded that "Particulate air pollution (i.e., particles small enough to be inhaled into the lung) is consistently and independently related to the most

serious [acute and chronic health] effects, including lung cancer and other cardiopulmonary mortality."[1] The press release accompanying the subsequent WHO 2006 guideline stated[2, 3]:

> "By reducing particulate matter pollution from 70 to 20 micrograms per cubic metre as set out in the new Guidelines, we estimate that we can cut deaths by around 15%," said Dr. Maria Neira, WHO Director of Public Health and the Environment. "By reducing air pollution levels, we can help countries to reduce the global burden of disease from respiratory infections, heart disease, and lung cancer which they otherwise would be facing."

The WHO statement continued:

> "These new guidelines have been established after a worldwide consultation with more than 80 leading scientists and are based on review of thousands of recent studies from all regions of the world. As such, they present the most widely agreed and up-to-date assessment of health effects of air pollution, recommending targets for air quality at which the health risks are significantly reduced."

And:

> "For example, in the European Union, the smallest particulate matter alone ($PM_{2.5}$) causes an estimated loss of statistical life expectancy of 8.6 months for the average European."

Hence, the WHO concluded not merely that the association of particles with early deaths is causal, but that the evidence is strong enough

to allow quantitative estimates of the mortality benefits of reducing particle concentrations.

A subsequent review of the literature was conducted in the United States in 2005-6. The Clean Air Act requires the Environmental Protection Agency (EPA) to review the National Ambient Air Quality Standards (NAAQS) every five years and determine whether revision is appropriate. As part of this process, the EPA is required to have its summary review of the science about each criteria air pollutant reviewed by an independent scientific review committee of outside experts, which is known as the Clean Air Science Advisory Committee (CASAC). In reviewing the 2005 EPA Staff Paper for particulate matter produced in the course of the NAAQS review begun in 1997, the CASAC stated, "[i]n summary, the epidemiologic evidence, supported by emerging mechanistic understanding, indicates adverse effects of $PM_{2.5}$ at current annual average levels below 15 $\mu g/m^3$."[4] In a 2006 letter regarding its recommendations for the final PM NAAQS, the CASAC reiterated[5]:

> *…there is clear and convincing scientific evidence that significant adverse human-health effects occur in response to short-term and chronic particulate matter exposures at and below 15 $\mu g/m^3$, the level of the current annual $PM_{2.5}$ standard.*

The CASAC emphasized:

> "Significantly, we wish to point out that the CASAC's recommendations were consistent with the mainstream scientific

> advice that EPA received from virtually every major medical association and public health organization that provided their input to the Agency, including the American Medical Association, the American Thoracic Society, the American Lung Association, the American Academy of Pediatrics, the American College of Cardiology, the American Heart Association, the American Cancer Society, the American Public Health Association, and the National Association of Local Boards of Health. Indeed, to our knowledge there is no science, medical or public health group that disagrees with this very important aspect of the CASAC's recommendations. EPA's recent "expert elicitation" study (Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between $PM_{2.5}$ Exposure and Mortality, September 21, 2006) only lends additional support to our conclusions concerning the adverse human health effects of $PM_{2.5}$."

As noted in the letter, the CASAC's conclusions are supported by all the major associations of health professionals, which include as members almost all researchers on heart disease, lung disease, and cancer. The American Medical Association, the American College of Cardiology, the American Heart Association, the American Thoracic Society, and the American Academy of Pediatrics all sent official letters to the EPA's

Administrator urging EPA to lower the $PM_{2.5}$ standard because of these known health effects.

In 2006, the World Health Organization also published the Global Burden of Disease report, which estimated that in the half of the world's population that lived in cities, particles were responsible for 811,000 early deaths per year (no particle exposure data was available for rural areas). A more recent update[6] included more recent literature and exposure estimates for rural areas, and concluded that particulate air pollution killed 3.2 million people in 2010 alone.

Hence, by 2006, every major scientific body involved in either research or the evaluation of research relating to particulate air pollution had concluded that it is a major health hazard whose consequences include early deaths. Since 2006, the evidence has become even more convincing.

The American Heart Association (AHA) appointed a panel of scientific experts to review new evidence on the risk posed by particles arising between the AHA's prior 2004 assessment and 2010. That review was published in Circulation, the world's leading peer-reviewed journal on heart disease. The abstract of that peer-reviewed paper summarizes the conclusions as follows[7]:

> "In 2004, the first American Heart Association scientific statement on 'Air Pollution and Cardiovascular Disease' concluded that exposure to particulate matter (PM) air pollution

contributes to cardiovascular morbidity and mortality. In the interim, numerous studies have expanded our understanding of this association and further elucidated the physiological and molecular mechanisms involved. The main objective of this updated American Heart Association scientific statement is to provide a comprehensive review of the new evidence linking PM exposure with cardiovascular disease, with a specific focus on highlighting the clinical implications for researchers and healthcare providers. The writing group also sought to provide expert consensus opinions on many aspects of the current state of science and updated suggestions for areas of future research. On the basis of the findings of this review, several new conclusions were reached, including the following: Exposure to PM <2.5 μm in diameter ($PM_{2.5}$) over a few hours to weeks can trigger cardiovascular disease–related mortality and nonfatal events; longer-term exposure (e.g., a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these

> findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality. This body of evidence has grown and been strengthened substantially since the first American Heart Association scientific statement was published. Finally, PM2.5 exposure is deemed a modifiable factor that contributes to cardiovascular morbidity and mortality. As already noted, in 2012 the Global Burden of Disease increased its estimate of particulate related deaths. "

In sum, one of the world's major medical societies found strong support that both short-term and long-term exposure to particles has significant impacts on health, including increased deaths.

EPA completed its most recent full review of the PM NAAQS in 2013, following several years of analysis. As part of this process, after extensive scientific review by the CASAC, the agency in 2009 published its Integrated Science Assessment (ISA) summarizing the state of the science about particulate air pollution. This ISA was particularly focused on examining the evidence for causality of the relation of particles with various health effects and drawing scientific consensus conclusions about that evidence.

It is useful to summarize the rigorous and extensive review that the ISA process entails. The process begins with EPA using internal scientists

and contracting with external, university scientists to write chapters of the ISA, which summarizes the state of the science about the air pollutant such as particles. Draft chapters are sent out for review by other external scientists and CASAC, and discussed at public meetings with CASAC, where others are encouraged to provide comments. Based on the review by CASAC, EPA revises the ISA and presents it for a second review. This process continues until the CASAC is satisfied and approves the ISA and its conclusions. EPA then drafts a Risk Assessment and a Policy Document. The Risk Assessment's goal is to quantify risk to the extent consistent with the CASAC review of the ISA. This risk assessment is then put through the same review protocol as the ISA and must be approved by the CASAC to be used. The Policy Document, which summarizes the policy relevant science and its implications for potential standards, likewise goes through the extensive review.

Returning to the 2009 particulate matter ISA, the EPA found in that assessment as follows:

> "Epidemiologic studies that examined the effect of $PM_{2.5}$ on cardiovascular emergency department (ED) visits and hospital admissions (HA) reported consistent positive associations (predominantly for ischemic heart disease [IHD] and congestive heart failure [CHF]), with the majority of studies reporting increases ranging from 0.5 to 3.4% per 10 $\mu g/m^3$ increase in $PM_{2.5}$. These effects were observed in study locations with mean 24-h avg $PM_{2.5}$ concentrations ranging from 7-18

μg/m^3 (Section 6.2.10) ...with effects becoming more precise and consistently positive in locations with mean $PM_{2.5}$ concentrations of 13 μg/m^3 and above (Figure 2-1). ...Toxicological studies have provided biologically plausible mechanisms (e.g., increased right ventricular pressure and diminished cardiac contractility) for the associations observed between $PM_{2.5}$ and CHF in epidemiologic studies. "

and:

"There is also a growing body of evidence from controlled human exposure and toxicological studies demonstrating $PM_{2.5}$-induced changes on heart rate variability (HRV) and markers of systemic oxidative stress (Section 6.2.1 and Section 6.2.9, respectively). Additional but inconsistent effects of $PM_{2.5}$ on blood pressure (BP), blood coagulation markers, and markers of systemic inflammation have also been reported across disciplines. ...Together, the collective evidence from epidemiologic, controlled human exposure, and toxicological studies is sufficient to conclude that a causal relationship exists between short-term exposures to PM and cardiovascular effects."

and:

"Collectively, the studies evaluated demonstrate a wide range of respiratory responses, and although results are not fully consistent and coherent across studies the evidence is sufficient to conclude that

> a causal relationship is likely to exist between short-term exposures to $PM_{2.5}$ and respiratory effects."

and:

> "An evaluation of the epidemiologic literature indicates consistent positive associations between short-term exposure to $PM_{2.5}$ and all-cause, cardiovascular-, and respiratory-related mortality (Section 6.5.2.2.). ... Collectively, the epidemiologic literature provides evidence that a causal relationship is likely to exist between short-term exposures to $PM_{2.5}$ and mortality."

and:

> "Evidence from toxicological studies provides biological plausibility and coherence with studies of short-term exposure and cardiovascular disease morbidity and mortality, as well as with studies that examined long-term exposure to $PM_{2.5}$ and cardiovascular mortality. Taken together, the evidence from epidemiologic and toxicological studies is sufficient to conclude that **a causal relationship exists between long-term exposures to PM2.5 and cardiovascular effects.**[8]

(Emphasis in original). Commenting on the ISA, the CASAC stated:

> "CASAC also supports EPA's changes to the causal determinations for long-term exposure to $PM_{2.5}$ and cardiovascular effects (from 'likely

> causal' to 'causal')" and "CASAC recommends 'upgrading' the causal classification for $PM_{2.5}$ and total mortality to 'causal' for both the short-term and long-term time frames."[9]

That is, the CASAC has concluded that the association between $PM_{2.5}$ and deaths is causal. Notably, the experts convened by the American Heart Association, the EPA authors, and the CASAC all agreed that there were convincing data on biological mechanism and broad coherence across epidemiology and toxicology.

More recently, the UK Royal College of Physicians issued a report in 2016 on the lifelong impact of exposure to air pollution stating that:

> "Population-based studies as well as modern biological science have revealed highly potent toxic effects of chronic exposure to 'modern-day pollutants', not only on the lungs but also on the heart and broader cardiovascular system. We are further recognising that the systemic effects of pollutants extend beyond the cardiopulmonary system to affect many other organs, increasing the risk of disease that begins from conception and persists across the lifecourse." (p vii)

And:

> "Each year, inhaling particulates causes around 29,000 deaths in the UK."(3), that "Infants living in areas with high levels of particulate air pollution are at increased risk of death during the first year of life, particularly from respiratory illnesses" (p42).

More recently, in 2013, the International Agency for Research on Cancer examined the evidence for the carcinogenicity of particulates. They had an expert panel review more than 1,000 peer-reviewed scientific papers and published their findings in a monograph (Vol 109, IARC monographs), declaring that particulate air pollution was a known human carcinogen. This finding was also published in a peer-reviewed journal.[10]

In 2015, Australia reduced its $PM_{2.5}$ standards to 8 $\mu g/m^3$ on an annual average, (compared to EPA's new 12 $\mu g/m^3$ annual standard) and 25 $\mu g/m^3$ for a daily limit, based on the overwhelming evidence of harm.

Similarly, there is a scientific consensus that there is no threshold for the effects of particles on deaths, indicating that any reduction in particle levels would result in lower death rates. For example, the CASAC has stated that "[a]lthough there is increasing uncertainty at lower levels, there is no evidence of a threshold (i.e., a level below which there is no risk for adverse health effects)."[11] The National Academy of Sciences concurs, stating "[f]or pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold."[12] This is also the view of the World Health Organization[13]. Recently, the Global Burden of Disease group estimated that air pollution is responsible for 5.5 million deaths worldwide, with a similar estimate recently published by the Lancet Global Health Commission[14, 15].

The World Health Organization's most recent assessments of the health impacts of air pollution states:

> "Ambient (outdoor air pollution) is a major cause of death and disease globally. The health effects range from increased hospital admissions and emergency room visits, to increased risk of premature death."

And:

> "An estimated 4.2 million premature deaths globally are linked to ambient air pollution, mainly from heart disease, stroke, chronic obstructive pulmonary disease, lung cancer, and acute respiratory infections in children."

WHO further explains that worldwide ambient air pollution accounts for:

- *25% of all deaths and disease from lung cancer*
- *17% of all deaths and disease from acute lower respiratory infection*
- *16% of all deaths from stroke*
- *15% of all deaths and disease from ischaemic heart disease [and]*
- *8% of all deaths and disease from chronic obstructive pulmonary disease*

WHO goes on to state specifically about $PM_{2.5}$, that "*[s]mall particulate pollution has health impacts even at very low concentrations – indeed no threshold has been identified below which no damage to health is observed*" (http://www.who.int/en/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health).

II.A.2. My Conclusions

The basis for this scientific consensus around causality and lack of a threshold is a large body of complementary scientific evidence that particulate air pollution is associated with early deaths, heart attacks, strokes, respiratory illness, lower birth weight, and decreased cognitive function. There are epidemiology studies demonstrating these associations, including ones using novel methods to eliminate the possibility that the effects seen are due to other causes. There are also animal studies showing that exposure to particles increases hardening of the arteries, destabilizes electrocardiograms and exacerbates pneumonia, as well as controlled human exposure studies that demonstrate exposure to particulate matter for a few hours increases inflammation and changes electro-encephalographic patterns in the brain. I summarize some of this literature in section III.

However, particulate air pollution is not merely fatally dangerous, it is ubiquitous. The satellite picture below shows a particle haze obscuring the view of most of the eastern coast of the United States, with patches of whiter clouds above. In contrast, at the lower left of the image, one can see an area that has escaped the particle haze, where the ground is clearly visible. Particulate air pollution is the only manmade object visible from space. The reason is that once airborne, particles can remain in the air for days, traveling with the wind, and exposing people far from where they were emitted.

The Only Manmade Object Visible from Space

In summary, I conclude, as does the scientific community, that any incremental increase in particle exposure, such as that caused by increased NOx emissions, will increase deaths in the United States, increase heart attacks and strokes, impair lung function, exacerbate asthma, and contribute to reduced cognitive function in children and accelerated cognitive decline in the elderly. +Details of the health studies that support my conclusions (and those of the medical associations, World Health Organization, the U.S. EPA, and Clean Air Scientific Advisory Committee) are found in subsequent sections.

II.B. Summary of Conclusions on Ozone Health Effects

Ozone pollution also has serious health effects, ranging from respiratory outcomes to mortality. Moreover, if there are any thresholds for these effects, they are so low (e.g., below 20 ppb) as to be inconsequential, since such levels are rarely seen in the US. Consequently, any incremental exposure to ozone causes incremental effects on health in the locations where that incremental exposure occurs. Moreover, this is not just my opinion; it is the scientific consensus, as reflected in the U.S. EPA's external Clean Air Scientific Advisory Committee's recommendations and review of EPA's Integrated Science assessment for ozone.

II.B.1. The Scientific Consensus

In its ISA[16], EPA states, with approval from the CASAC:

"The 2006 O3 AQCD concluded that there was clear, consistent evidence of a causal relationship between short-term exposure to O3 and respiratory health effects. This causal association is more substantiated now by the effects observed across recent controlled human exposure, epidemiologic, and toxicological studies indicating associations between short-term O3 exposures and a range of respiratory health endpoints from respiratory tract inflammation to respiratory-related emergency department (ED) visits and hospital admissions. Short-term O3 exposures induced (or were associated with) statistically significant declines in lung function. An equally

> strong body of evidence from controlled human exposure and toxicological studies demonstrated reversible O3-induced increases in inflammatory responses, epithelial permeability, and airway hyperresponsiveness that were found to last for 18-24 hours after O3 exposure. Toxicological studies in animals provided additional evidence for O3-induced impairment of host defenses. Combined, these findings from experimental studies provided support for epidemiologic evidence, in which short-term increases in O3 concentration were consistently associated with increases in respiratory symptoms and asthma medication use in children with asthma, respiratory-related hospital admissions, and ED visits for chronic obstructive pulmonary disease (COPD) and asthma. Additionally, recent epidemiologic evidence supports the range of respiratory effects induced by O3 by demonstrating that short-term increases in ambient O3 concentrations can lead to respiratory mortality. The combined evidence from these disciplines supports the conclusion that **there is a causal relationship between short-term O3 exposure and respiratory effects**." (emphasis in original).

This review was subject to outside peer review by EPA's expert Clean Air Scientific Advisory Committee. Moreover, other national and international organizations have supported similar conclusions. For example, the World Health Organization in 2005 set an eight hour maximum

ozone standard of 100 μg/m^3 (50ppb), which is considerably lower than the recently lowered EPA standard of 70ppb[2]. In doing so, they stated:

> "Significant additions to the health effects evidence base have, however, come from epidemiological time-series studies. Collectively these studies have revealed positive, small, though convincing associations between daily mortality and ozone levels, which are independent of the effects of particulate matter. Similar associations have been observed in both North America and Europe. These latest time-series studies have shown health effects at ozone concentrations below the previous guideline of 120 μg/m^3 (60 ppb) but without clear evidence of a threshold."

More recently, the Global Burden of Disease group concluded that ozone caused increased respiratory disease mortality, and estimated the worldwide burden as 152,000 deaths per year in 2010[14]. The WHO HRAPIE project convened scientific experts to assess the state of the art on air pollution, and concluded that increased summer daytime ozone concentrations increased annual death rates[17].

II.B.2 My Conclusions

I conclude that short-term exposure to ozone produces increases in daily death rates, hospital admissions for respiratory disease, and other health outcomes. I also conclude, based on the most recent studies, that

long-term exposure to ozone increases annual death rates. The basis for these conclusions is discussed in section III.

II.C. SUMMARY OF NOX HEALTH EFFECTS

The 2016 ISA for NO2 concludes that there is a causal association between NO2 and respiratory hospital admissions. It also describes additional health effects that are likely causal. The World Health Organization has taken a stronger position, concluding that there is a causal effect on both respiratory hospital admissions, and daily death rates. However, a number of important studies not considered in either document have since been published. They indicate stronger evidence for the acute effects on daily deaths and hospital admissions, but also strong evidence for an effect of annual average NO2 on annual death rates. This evidence is discussed in section III.

III. HEALTH EFFECTS OF PARTICLES, OZONE, and NOX

III.A.1. PARTICULATE AIR POLLUTION KILLS PEOPLE

Multiple types of studies have been used to examine the relationship between particles and mortality: long-term exposure studies have focused on the association between longer-term exposure to particles and life expectancy, whereas other studies have looked at the acute responses within a few days of exposure. Finally, there are some intermediary studies that attempt to bridge the gap. Within studies of longer-term

exposure there are further distinctions based on whether the differences in exposure among people is just between cities, or whether it includes, or is limited to, contrasts within a metropolitan area. Also, some studies incorporate changes over time in exposure. I discuss these various types of studies below.

III.A.1.a. Long-Term Exposure to Particles Increases Long-Term Death Rates

I find, as have the major scientific organizations, that there is clear, convincing evidence that exposure to particles shortens life expectancy by substantial amounts. I base this judgment on the extensive literature, as discussed below.

In 1970, Lave and Seskin published a paper analyzing the relationship between age-standardized mortality rates in U.S. cities and average particle concentrations in those cities.[18] The advantage of that study was that the mortality experience of the entire population of each city was compared to the average particle concentration from the population-oriented monitors in the city. The difficulty was that no individual level covariates (i.e., other individual factors such as hypertension, diabetes, smoking, etc. that may differ on average across the people in different cities and might explain the differences among those cities in mortality rates) were controlled, raising questions about confounding (i.e., that another variable explains the observed association).

More recent studies have alleviated that problem by recruiting individuals in various areas, measuring individual covariates in each person, and following those people over time. These are referred to as cohort studies. It is these new cohort studies, starting with the Harvard Six City Study, and including the American Cancer Society (ACS) study, the Women's Health Initiative study, the Nurse's Health Study, the ESCAPE study, etc., together with parallel findings for short-term effects of particles on deaths, and toxicology results that confirm the mechanisms, that led the CASAC to tell EPA to conclude that the association of particles with total mortality was causal. As noted above, the 2009 EPA particulate matter ISA concluded that a causal relationship exists between both short-term and long-term exposures to $PM_{2.5}$ and mortality.

Below, I summarize the studies that support this conclusion. In doing so, I emphasize addressing a set of issues that have been raised by critics in comments to EPA and the CASAC. Those critiques were rejected as not valid by both organizations, as well as by WHO and medical societies, for reasons discussed below. The key issues that have been raised, and refuted in my opinion, are measurement error, confounding, and lack of biological plausibility.

Measurement Error

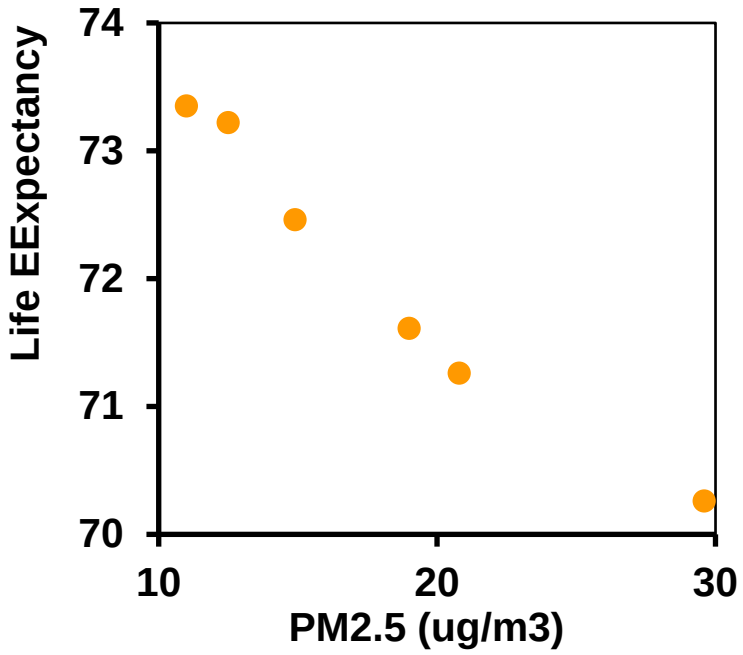
Ideally, when looking at whether people with higher exposure to particulate air pollution suffer worse health outcomes, we want to assess

that exposure as accurately as possible. Differences between the actual exposure of an individual and what was used as exposure in a study are referred to as measurement error in exposure. Over time, better methods of exposure assessment have allowed studies to reduce that error.

Early studies reporting associations between particulate air pollution in each city or neighborhood and mortality rates in those locations were criticized by industry because of the potential measurement error in assigning citywide average particle concentrations as the exposure estimate to everyone in a city. While this certainly does produce measurement error in the exposure estimate, it is important to realize that the principle effect of such error is to *reduce* the estimated size of the effect of particles on death rates and widen the confidence bands about that estimate[19]. Heuristically, this can be understood as follows. Suppose particles in the air are correlated with the risk of dying, but I measure the particle exposure of people with error. Hence some of the variation in the particle exposure we use in our study is real, and some is noise; variation due to our error in estimating people's exposure. Presumably, this noise is not correlated with that person's risk of dying, and so by using the noisy exposure variable, the overall correlation with mortality is reduced below what it would have been had the true exposure been used. Hence, exposure error is unlikely to explain the finding of a statistically significant effect in a cohort study or result in an overestimate of the effect.

Regardless, reducing exposure error has been a major focus of more recent studies. For example, a key advantage of the Six City Cohort Study over other early cohorts is the study's smaller error in exposure.[20] The Six City Study chose a neighborhood within each city, recruited a random sample of people from that neighborhood, and put a population-oriented particle monitor in the middle of each neighborhood. In the Six City Study, most subjects lived within a few kilometers of that central monitor, and thus the exposure error was much lower than in studies, such as the American Cancer Society (ACS) cohort, that used the average of all monitors in a greater metropolitan area as the exposure for all participants living in that city. The Six City Study found a substantial reduction in life expectancy with higher exposure to particles. Moreover the size of that reduction was larger than the one found in the ACS study. This higher effect estimate is what we expect from a study with less exposure error. This magnitude of the impact on life expectancy is indicated in the figure below, which shows the life expectancy in each city, after adjusting for age, sex, cigarette smoking, occupation, education, obesity, and chronic disease, plotted against the mean $PM_{2.5}$ in that city. To put the 3-year difference in life expectancy between low and highly polluted communities in perspective, between 1995 and 2005 life expectancy in the U.S. increased by 2 years. Hence, exposure to $PM_{2.5}$ can obliterate the effects of one and a half decades of medical progress on life expectancy.

Figure 1. Life expectancy decrease with increasing $PM_{2.5}$ in Six City Study



Further evidence that reducing exposure error tends to increase estimates of the effects of particles on deaths comes from a number of more recent studies. For example, a study examined the 22,905 participants of the American Cancer Society study living in Southern California using a geographic information system-based exposure model, which captures the local exposure gradient within Southern California. This study reported much larger effect size estimates for $PM_{2.5}$ than the original study.[21] Similarly, the Women's Health Initiative study found a larger effect on mortality when they used more local, within-city exposure estimates than when they just used exposure differences between cities.[22]

Another cohort study examined over 66,000 nurses living in the Northeast and upper Midwest.[23] We used a spatial model that estimated individual exposures at the *home address* of each nurse, and found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ at a nurse's address was associated with a 26 percent increase in risk of dying in that year. As with other studies with individualized exposure estimates based on residential location, this increase was considerably larger than the increase seen in the American Cancer Society study, which only looked at exposure differences across cities. We also found that this increase was predominantly seen within a year of the change of exposure. Again, reduced error in estimating individual exposure resulted in higher estimates of the effects of particles on mortality.

There are also specific methods to correct for measurement error that have been applied. For example, Hart and coworkers have applied the measurement error correction estimation methods developed by Professor Spiegelman, a world expert in the subject[24]. When they used validation studies, which measured both personal $PM_{2.5}$ monitoring and the modeled exposure to examine the nature of the exposure error, and adjusted for that error in the Nurses Health Study Cohort, they found that the measurement error corrected slope was higher than the original one, not lower[24]. She further applied this approach in a different cohort, the Netherlands Cohort, to look at the association of $PM_{2.5}$ with lung cancer.

Again, correcting for the measurement error increased the slope (the change in rate per unit change in $PM_{2.5}$)[25].

More recently, we performed a meta-analysis of 53 cohort studies of PM2.5 and mortality and looked at what characteristics of the studies predicted the concentration-response slope. We found that studies with more exposure error had smaller slopes than studies with less error in exposure[26].

Hence, the use of more localized measures of exposure, with resultant lower exposure error, or of measurement error correction, generally has resulted in larger effect estimates.

Confounding

Confounding occurs when the difference in exposure across study subjects is correlated with differences in some other predictor of mortality, which may be an alternative explanation for the difference in health seen in people with whose exposures differ. Importantly, to be a confounder, a characteristic must be correlated with both the health outcome being studied, **and** with the exposure being studied. Only then can exposure act as a surrogate for the confounder in predicting health. For example, the APOE4 genotype is a risk factor for faster cognitive decline in the elderly. However, it is not a confounder of air pollution, since it is not correlated with exposure to pollution. Hence, an association between air pollution and health cannot result from people with the APOE4

genotype having higher exposure (since they don't). Thus, critiques that cite predictors of health that were not measured and controlled for in air pollution studies as a reason to doubt the validity of those studies have little meaning unless they provide evidence that those predictors of health are also predictors of particulate air pollution.

The obvious solution to the presence of a confounder is to control for that other predictor of mortality, and only look at exposure differences that are independent of it (or them). Cohort studies all do this by collecting information on the other predictors, such as diabetes, smoking, etc., that are known to increase the risk of dying. In general, cohort studies looking at particulate matter find very little evidence for confounding—that is, the effect of particulate air pollution changes little with or without control. Indeed, in one analysis of the American Cancer Society study, Pope showed that he obtained the same result from his cohort study as if he had used the approach of Lave and Seskin and controlled for nothing.

Equally important, the Six City Study went further, and showed the association of air pollution with life expectancy before and after controlling for each potential confounder, such as smoking, hypertension, diabetes, occupational exposures, obesity, etc. There was no evidence of confounding by any of the covariates examined except age. A recent paper of Di looked at the correlation between smoking and body mass index (a measure of obesity) with $PM_{2.5}$ in a representative sample of over 55,000

Medicare beneficiaries, and reported no association of either variable with $PM_{2.5}$[27]. Hence, these variables are not plausible confounders.

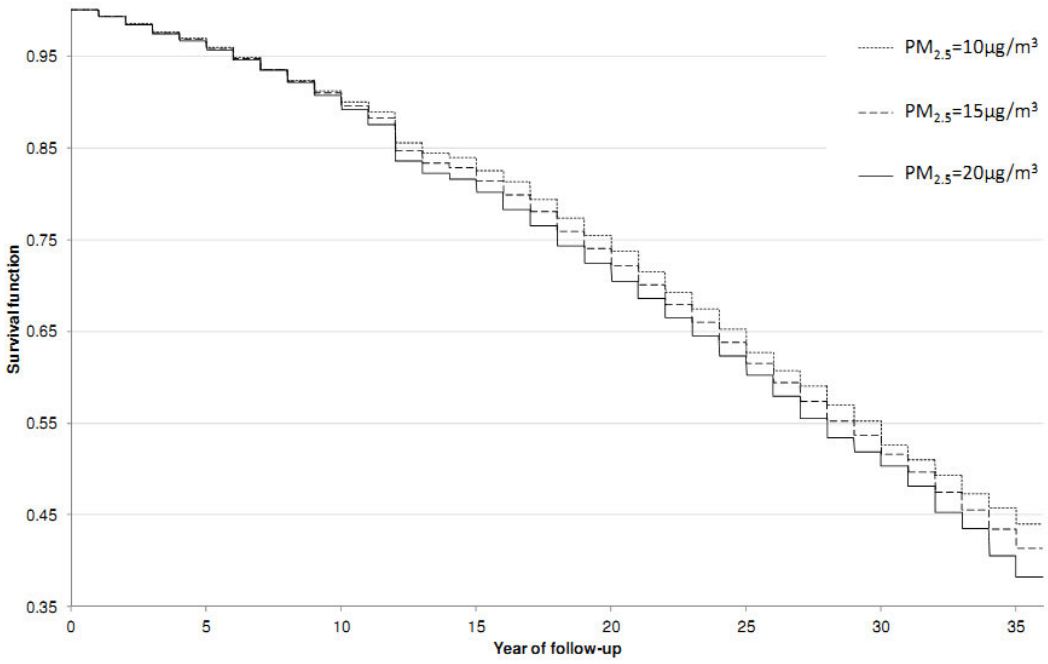
Another recent analysis, which extended the previous analyses of the American Cancer Society study to include more years and more data, additionally controlled for census level data on socioeconomic status, based on where the participants lived.[28] Interestingly, they found that control for neighborhood socio-economic status increased the risk associated with particles, rather than decreasing it. Another examination of this issue was conducted by Brochu[29]. He specifically examined the question of whether $PM_{2.5}$ was correlated with socio-economic status, and found that on a large spatial scale, on the order of multi-state regions, there was a correlation between $PM_{2.5}$ and socio-economic status. However, as one narrowed one's focus to states, or to variations within states or urban areas, the correlation disappeared, and with it, the potential for it to be a confounder.

Other critiques of these studies have argued that they may not have controlled well enough for smoking or other confounders because they were proportionate hazard models. What this means is that the studies assume that the effect of, e.g., smoking is the same in each year of follow-up. However, a re-analysis by Burnett of the American Cancer Society study let the effects of smoking and all other confounders vary over time and found no change in the effect of particles. Lepeule and coworkers re-analyzed the Six City Study with 11 additional years of follow-up.[30] We specifically

addressed this question by letting the effect of smoking, education and sex on the risk of death differ for each of the 35 years of follow-up. There was no change in the effect of $PM_{2.5}$ on deaths from doing so.

We then went further and let the effect of $PM_{2.5}$ on deaths differ in each year of the 35-year follow-up. The figure shown below demonstrates that even under this scenario, we consistently find a higher proportion of people surviving at 10 µg/m^3 compared to higher exposures, with an additional 5% of the population surviving for 35 years after recruitment if their exposure were 10 µg/m^3. Put another way, the proportion of a random population sample expected to be alive 35 years after recruitment would be 20% higher in a city with PM concentrations of 10 µg/m^3 rather than a city with 20 µg/m^3.

Figure 2. Percent of the Population Surviving vs. number of years under study in the Six City Study at three different concentrations of $PM_{2.5}$.



These results provide reassurance that confounding is unlikely. It is also important to look at multiple studies and multiple study designs that have different potentials for such confounding to gain confidence that there is no confounding regarding particle pollution and health effects.

For example, suppose there is an unmeasured health risk (say, diet) in a cohort study that predicts mortality. For this to be a problem in the traditional analysis contrasting mortality rates across cities with air pollution across cities, dietary differences across cities would have to be correlated with particulate air pollution levels across cities. It is

unclear why this would happen, but suppose this were true in one study. Unless there is a *systematic process* that increases high-fat diet in towns with higher pollution, another cohort study is unlikely to find the same problem.

Again, suppose, for some reason, there were such a systematic correlation of air pollution across cities with dietary differences across cities. What then, of the studies noted above, contrasted air pollution differences across neighborhoods within cities with mortality rates by neighborhoods within cities? It seems unlikely that the same correlation would persist within city. And what about the studies that estimate exposure at the address of each participant of the cohort? Hence, the ISA and all medical groups reviewing the literature on particulate air pollution looked for—and found—consistency across such different studies and study types.

More recently, several studies have used additional methods to assure that the observed association between particle pollution and health is causal.

Causal Modeling

To understand what is generally called causal modeling, it is useful to consider why we believe that a randomized control trial produces a causal estimate of effect. Because the population is randomly divided into two groups, one assigned treatment and the other a placebo, the

distribution of other predictors of the health outcome being studied should be the same in both groups. Treatment is *independent* of those other predictors and therefore cannot be confounded by them. Hence, if we see a difference in health between the two groups, we believe it is causal.

Such randomized trials for long-term exposure to air pollution are essentially impossible. The field of causal modeling in epidemiology and statistics seeks to find other approaches to achieve the same result: make exposure independent of the other predictors of the health outcome, so confounding does not exist. There are two basic approaches. One seeks to isolate a part of the variation in exposure that is independent of confounders, including unmeasured ones, by focusing on an intervention, a change, or a variable that is responsible for some change in exposure, but is not plausibly associated with other predictors of the health outcome. This can include natural experiments, where an abrupt change in exposure occurs due to some factor not otherwise expected to produce the health effect studied, or attempts to remove systematic trends over time in health and exposure and examine whether the random fluctuations in health and exposure about those trends coincide, etc.

The second approach seeks to reweight the population being studied so as to ensure that exposure is independent of what might otherwise be a confounder. For example, suppose that people with higher cholesterol levels tended to have higher air pollution exposure. In that case, exposure is not independent of cholesterol, and confounding is possible.

Nevertheless, there will be some people in the study population with high cholesterol but low exposure. There just aren't enough of them to balance out the larger number of people with high cholesterol and high exposure. However, if we, for example, counted those people with high cholesterol and low exposure twice in our analysis (or whatever the appropriate multiplier is to balance out the number of people who are high in cholesterol and exposure), we can produce a new analysis population where pollution is independent of cholesterol. Both of these approaches to causal modeling have been applied in the study of long-term exposure to $PM_{2.5}$ and death.

Studies that examine change in exposure play an important role in understanding the effects of particles for several reasons. First, if particle-induced changes in health are permanent and we must wait for a new generation before seeing public health improvements follow the exposure reductions, there are important public health implications. Second, showing that a change in exposure produces a change in response more directly addresses the causality of the association. If A causes B, then changing A will change B. Finally, cross-area comparisons between lung function, mortality rates, or any other health response and cross-area differences in exposure must be concerned about confounding by predictors of outcome that vary geographically. Naturally, epidemiology studies try to identify such variables and control for them, as noted above.

Examinations of year-to-year changes in exposure **within** a location and year-to-year changes in mortality rates do not suffer the potential confounding that, as above, some unmeasured confounder may differ from one city to another, or even from neighborhood to neighborhood within a city, in parallel with air pollution differences. These factors cannot be correlated with exposure that only varies from year-to-year *within* a city or neighborhood. A different set of variables, which do vary from year-to-year, might confound the air pollution association, but they are fewer and different from the potential confounders of the cross-sectional associations. Consequently, it is implausible to expect them to confound by the same amount. Hence, if similar associations are seen using this very different study design as well, it provides greater confidence that the associations are causal. Moreover, if long-term time trends in air pollution and mortality rates are removed, and we focus only on year-to-year differences in $PM_{2.5}$ around its location specific trend, then this exposure can neither be confounded by factors that vary from city to city, nor by long term characteristics of the participants in the study (cumulative smoking, obesity, etc.) that change slowly over time. Importantly, the exposure variation we study (year-to-year differences around location specific trend) is also independent of predictors of health we have not measured. This approach is discussed further below.

Consequently, a key finding for cohort studies of mortality has come from studies examining *changes* in exposure and *changes* in mortality rates.

Most of the cohort studies, including the original Six City Study, have contrasted a measure of long-term exposure with long-term survival. They tell us that people have shorter lives in more polluted cities or neighborhoods. They do not directly tell us what mortality reduction accompanies a reduction in exposure. In a follow-up of the Harvard Six City Study, Laden and coworkers provided precisely that estimate.[31] We examined a further 10 years of follow-up and mortality in the six cities. In some cities there was a substantial drop in pollution between the first and second follow-up periods, in some cities there was a moderate drop, and in some cities there was little or no change. The mortality rates followed the same pattern: where there was a substantial drop in pollution, there was a substantial improvement in life expectancy; where there was little change in pollution concentrations, there was little change in life expectancy. The slope for *change* in exposure and *change* in death rate was similar to, but slightly higher than, the original cross-sectional slope.

If the mortality rates change *within* a town as the air pollution changes, and those changes fit on the same dose-response curve as the original curve looking at mortality and pollution *across* towns, this provides substantial assurance that the association is not confounded, because the factors that are likely to confound an association of temporal change are usually different from those that might confound a cross-sectional study, and there is no reason for the confounding of two

different estimates by different confounders to produce similarly sized estimated effects for particles.

This conclusion is also supported by natural experiments, the abrupt changes I mentioned above. Pope and coworkers reported that mortality fell in the Utah Valley in the year a strike closed a steel mill, and returned to its previous level the next year when mill operations resumed.[32]

The finding of a rapid change in mortality risk associated with change in particle exposure fits nicely with a similar report for lung function (a predictor of life expectancy) described in a later section about the Swiss Cohort Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) study.[33] In that study, we looked at lung function decline in adults (lung function falls from approximately age 35 continuously with age). We estimated $PM_{2.5}$ concentrations at each participant's home address and found that the rate at which lung function fell in adults depended on the rate at which air pollution fell at the address where they lived. If particulate air pollution fell by more at a participant's address, the rate of lung function decline was slower.

Another study examined changes in life expectancy across 51 metropolitan areas in the United States, between 1980 and 2000. The study found that 15 percent of the *increase* in life expectancy during that period came from *decreases* in air pollution, and that in the more polluted cities that cleaned up, life expectancy was increased by 10 months.[28]

Zanobetti and Schwartz followed a cohort of over 190,000 subjects discharged alive from hospitals following myocardial infarctions (heart attacks).[34] We used a form of causal modeling called differences in differences. We analyzed whether year-to-year fluctuations in air pollution within a city about its time trend were associated with year-to-year differences in mortality rates in all Medicare participants in that city around their time trend, controlling for age, race, and sex of each participant.

Using this approach, no predictors of mortality rates between cities can be confounders, because the analysis is only within city, and hence exposure is not associated with those variables. No time trends in city specific predictors of mortality rates can be confounders, because time trends are removed from air pollution and therefore it cannot be correlated with those predictors. What we are looking at is whether random year-to-year variations about its time trend in pollution in a city predict similar year-to-year variations in mortality rates around their time trend. Since year-to-year fluctuation in air pollution concentrations around their long-term trends are driven by things like year-to-year changes in what fraction of the time the wind was blowing from more polluted or less polluted upwind regions, and year-to-year variations in wind speed and inversions, it is difficult to imagine it is correlated with other factors influencing mortality rates. Specifically, we looked at year-to-year changes in exposure **within** 21 cities and how they related to

the probability of surviving that year. We adjusted for long-term time trend, and did separate analyses within each of 21 cities. We then combined the results across cities. We reported a significant association with PM_{10} in this susceptible subgroup and larger coefficients (the slope between exposure and mortality risk) than were seen in the Six City Study. A follow-up study looking at people with chronic bronchitis and emphysema in the same manner found a similar result.[35] This is a generalization to multiple locations and time periods of a well-known causal modeling technique called *differences in differences*.

Another study in a similar vein was the work of Janke and coworkers.[36] They looked at 354 local governmental units in England, comparing the annual mortality rates for multiple years in each location to the annual air pollution concentrations, controlling for *location and local time trend*. Again, they were looking at whether random deviations from year-to-year in air pollution around the local means and local time trend are correlated with random deviations in mortality rates around the local mean and local time trend. Such a design leaves little room for confounding. They found a strong association with particulate air pollution.

Subsequently, Kioumourtzoglou and coworkers followed 35 million Medicare participants in 207 cities for 11 years using the same differences in differences approach.[37] The results confirmed a causal association between particulate air pollution and annual mortality rates.

Specifically, we found that a 10 μg/m^3 increase in $PM_{2.5}$ produced a 19 percent increase in mortality rates in the 35 million study subjects.

More recently, Abu Awad and coworkers examined over 12 million Medicare beneficiaries all over the US who moved to a new ZIP code[38]. Since the participants are unlikely to know the air pollution concentrations at different potential addresses they considered moving to, and we only compared people who moved from the same original neighborhood, we believe that the change in pollution from old to new ZIP code is likely independent of any predictors of mortality. We checked this by examining how the distribution of age, eligibility for Medicaid, median income, median house value, smoking rates, previous hospitalization for a heart attack, etc. compared with the changed in air pollution, and found there was no difference. To further assure a causal estimate, we only compared individuals who had moved from the same ZIP code to each other. Hence median income, green space, past air pollution exposure or any other characteristic of the old neighborhood were controlled for. We further controlled for socio-economic and other factors at the new ZIP code. We found a significant association of $PM_{2.5}$ with mortality, with a larger impact when we restricted to only ZIP codes with $PM_{2.5}$ concentrations below the current ambient standard of 12 μg/m^3.

In addition, Yitshak-Sade and coworkers did a differences in differences analysis of all Medicare deaths (5.8 million) among Medicare participants (15.4 million) in the Northeast and Mid-Atlantic states,

using ZIP code level rates, they again reported a significant association with PM2.5[39]. Similarly, a recent differences in differences analysis in central Italy (including Rome) reported a significant association of particles with annual deaths[40].

Using another causal modeling approach, Wang and coworkers looked at all Medicare participants in the Southeast and reported that $PM_{2.5}$ increased death rates in that population[41]. The approach is called doubly robust, because if either the analysis of mortality vs. predictors is done correctly or the analysis of $PM_{2.5}$ vs. predictors is done correctly, an unbiased causal estimate is produced.

Another study of Pope and coworkers again looked at a natural experiment[42]. A copper smelter strike in the Southwest between 15 July 1967 and early April 1968 shut down all the smelters in the region. During the 1960s, smelters accounted for a substantial fraction of all particles in these southwestern states. As reported by Trijonis and Yuan (1978) and Trijonis (1979) this strike led to significant reductions in particles in the Southwest of approximately 2.5 $\mu g/m^3$ in mass concentration during the strike, with the concentrations going back up after it was settled. This natural experiment really is equivalent to a randomized trial. The population of the downwind states had no choice in the matter—they were exposed to higher, lower, and then higher particle concentrations over time, just as in a crossover trial for a drug. Nor did they even have a

perception that their exposure had changed, since there was little public attention to air pollution in this period.

Pope and coworkers analyzed this natural experiment to see how mortality rates change in response to the change in particle concentrations. After controlling for time trends, mortality counts in bordering states, and influenza/pneumonia deaths, they found that the 2.5 $\mu g/m^3$ decrease in particle concentrations resulted in a 2.5% decrease in the number of deaths in the four-state region. This unambiguously establishes particles as a cause of early death.

In comparison, a 2.5 $\mu g/m^3$ decrease in long-term average $PM_{2.5}$ concentrations in the American Cancer Society Cohort study was associated with about a 1.5% decrease in deaths, whereas in the Harvard Six City Cohort, the same decrease was associated with a 4% reduction in deaths. So these results from a natural experiment are similar to those from cohort studies. Moreover the exposure change during the strike only lasted for 8 months, so the impact of a change for a year or more would likely have been higher. Hence this natural experiment not only shows that particles kill people, its effect size is consistent with the long-term studies of mortality from following cohorts.

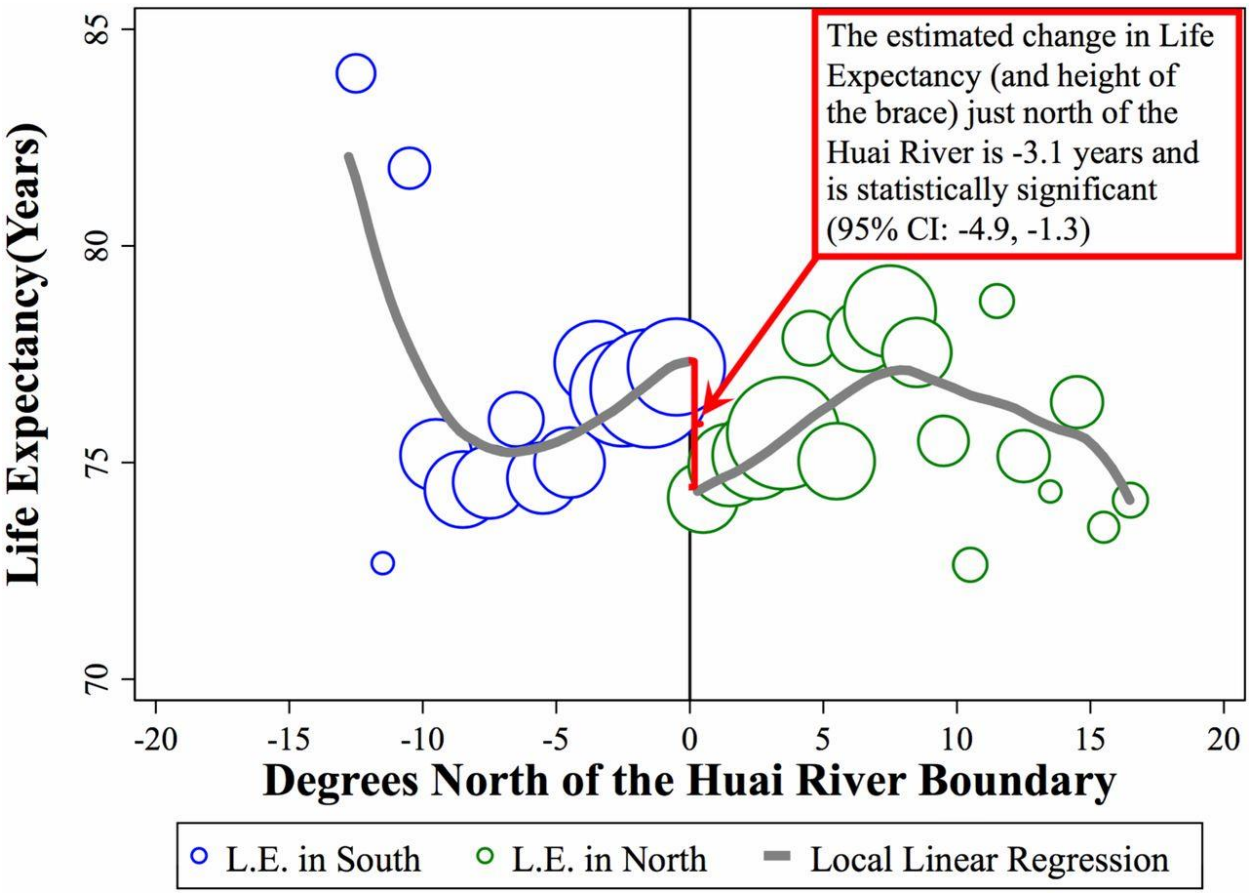
Wang applied the differences in differences approach to the entire population of New Jersey[43]. We identified the census tracts of all deaths in New Jersey over a six-year period. We then controlled for all ~2,000 census tracts in the state, so there could be no confounding by any factor

that differed among census tracts, and for each year of the study, so that differences in time from year-to-year could not confound the association. We used satellite remote sensing to measure $PM_{2.5}$ concentrations in each year in each census tract and examined whether fluctuations within census tract in $PM_{2.5}$ concentrations around the tract mean and statewide trend were associated with fluctuations in the mortality rate within each census tract around its tract mean and the statewide trend. We found a causal estimate of a 15% increase in annual deaths for each 10 $\mu g/m^3$ increase in $PM_{2.5}$.

Other studies have taken the alternative approach I mentioned, weighting the study population using what is called a propensity score to make exposure to $PM_{2.5}$ independent of all measured confounders. Wang applied this to a study 13 million Medicare participants in the Southeast U.S. He found a significant causal effect of $PM_{2.5}$ on deaths in this population, indicating that each 1 $\mu g/m^3$ increase in $PM_{2.5}$ in the Southeast resulted in an additional 5,000 deaths per year in the Southeast Medicare population.

Another quasi-experimental study used a causal modeling approach called a regression discontinuity design. Instead of looking at a sudden change in exposure with time, this examined a sudden change spatially. China had a policy of providing subsidized coal for heating to people living in the north of the country, but not in the south where it was warmer. Necessarily, they needed to define a boundary where the subsidy

stopped. The regression discontinuity design says that if one looks at the air pollution concentrations in China, going from the south to the north, one would expect this policy to cause a discontinuous change, with a sudden jump in concentration at the boundary. If air pollution increases mortality rates, then one would similarly expect a discontinuous jump in the mortality rate at the border. This is precisely what the investigators found. To ensure that differences in other factors between north and south China were not confounders, they did not simply contrast the averages between north and south, but rather let the mortality rate vary smoothly approaching the boundary from the north and from the south and found a discontinuous jump at the boundary. That difference is a causal estimate of the effect of the pollution, because in the immediate vicinity of the line, it was an arbitrary decision as to which side of it people lived, and hence exposure was randomly assigned. The investigators found that there was more particle pollution north of the line, and that pollution caused more than a 5 year loss of life expectancy[44]. A follow-up study looked at more recent years with lower exposure, and continued to report a significant 3.1 year drop in life expectancy and increase in particle concentrations occurred at the boundary line[45]. The below figure from their paper shows life expectancy rising as one moves from 10 degrees south of the boundary to the boundary and rising at a similar rate north of the boundary for another 10 degrees, but with the line shifted down noticeably.



Recent Additional Studies and Consistency

Studies of long-term effects of particles on mortality rates in large cohorts continue to be produced and find similar results. The ESCAPE study pooled 19 cohort studies in Europe and reported a significant association between $PM_{2.5}$ and mortality rates, with a percent increase in death rates per 10 µg/m^3 of $PM_{2.5}$ almost identical to the Six City Study results[46]. A study of 121,000 nurses by Hart reported slightly larger increases in mortality rates for the same increment in exposure[24]. They also demonstrated that error in estimating exposure resulted in an underestimate of the true effect. In 2015, a study which followed over 7

million adults in the Netherlands reported an association between PM_{10} and mortality, after controlling for both individual level and area level socioeconomic status. Converted to $PM_{2.5}$, the estimated size of effect was identical to that Lepeule reported in the Six City Study[47]. A study of a Canadian cohort of 2.5 million participants reported a significant association with $PM_{2.5}$ concentrations. A cohort of over 120,000 participants followed for lung cancer incidence reported a significant association with $PM_{2.5}$[25]. And a further analysis of the ACS cohort using better exposure modeling, longer follow-up, and twice as many deaths, reported that both local and regional $PM_{2.5}$ were associated with increased risks of death[25, 48, 49]. The National English Cohort study reported a significant and somewhat larger effect of $PM_{2.5}$[50].

Most recently Di followed the entire Medicare population of the contiguous U.S. (61 million people) for up to 13 years, using $PM_{2.5}$ exposure at their ZIP codes of residence. He reported a strong association between $PM_{2.5}$ and mortality rates, with no evidence of a threshold[27].

In addition, I published a paper, using a causal modeling approach (inverse probability weighting) that directly analyzed the effect of $PM_{2.5}$ on life expectancy instead of mortality rate[51]. I found that compared to a population exposed to 12 $\mu g/m^3$, the same population exposed to 7.5 $\mu g/m^3$ would have a 0.89 year longer average life expectancy.

It is important to note that these epidemiology studies are supported by toxicology studies, described in more detail below, which show that

particle exposure can destabilize atherosclerotic plaque (which increases the risk of a heart attack or stroke), can promote the growth of atherosclerosis (hardening of the arteries), can increase inflammation (which is a risk factor for heart attacks and strokes), disturb electrocardiogram rhythms (which is a risk factor for arrhythmias and heart attacks), can increase factors that promote blood clotting (a risk factor for heart attacks and strokes), can alter expression of hundreds of genes related to inflammation and other stresses in cells, etc. There are even short-term randomized trials placing particle filters or sham filters in people's homes showing that real filters lower blood pressure, reduce insulin resistance, stabilize electrical control of the heart, reduce systemic inflammation, and lower stress hormones in the body. There can be no question as to the causality of those effects. Hence, there is no doubt that particles cause heart disease and increase the risk of dying from it.

The association between airborne particles and mortality implies a very large public health impact. For example, the Laden paper suggests that emission reductions that produced an average 5 $\mu g/m^3$ decrease in $PM_{2.5}$ concentrations in the United States would be associated with a 5 to 10 percent decrease in total mortality, which is 100,000 to 200,000 fewer deaths per year. **For comparison, the lower bound estimate is more deaths than from AIDS, breast cancer, and prostate cancer combined.** Indeed, in another comparison, the Lancet Global Health Commission reported that globally, air pollution kills more people than AIDS, Malaria, and

Tuberculosis. In contrast to those diseases, we know how to lower $PM_{2.5}$, and hence cure the particle associated deaths. Lowering NO_2 emissions is part of that cure.

III.A.1.b. Short-Term Exposure: Particles and Mortality

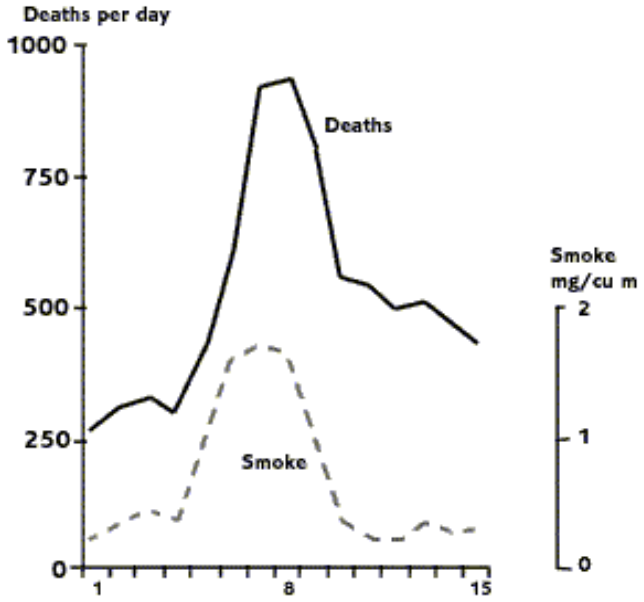
It is my opinion that airborne particles clearly are associated with increased deaths on that day and the next few days. These results are confirmed by large numbers of studies.

The earliest and clearest studies of the effects of short-term changes in particles on mortality focused on severe air pollution episodes. Death counts for several days or weeks during and immediately after the episodes were compared to those before and after the event, or to the same dates in other years. These studies unambiguously showed that high concentrations of PM could increase death rates.

For example, the figure below shows daily deaths and daily black smoke concentrations in London in the 1952 episode.[52] As the month began, a combination of weather fronts produced a sharp drop in wind speed, particularly over the southeast part of the country. On December 4, the wind velocity at the Kew Observatory in west London dropped from 6 knots at noon to 0 knots by 6 p.m. A low-level thermal inversion combined with this to produce a rapid increase in particle concentrations. Deaths rose rapidly with the increase in particles. They also trailed off rapidly with the fall in particles. However, while there was an initial rapid decline,

it had a slow tailing off and did not drop back to pre-episode levels immediately. Instead it remained somewhat elevated for several weeks. This long tail of increased deaths after an episode has been reported in other sudden air pollution episodes, and is consistent with the results of Zanobetti et al.,[53] where much lower particle exposures were shown to have an effect on mortality for weeks after the exposure.

Figure 3. Daily deaths and smoke concentrations in London in 1952

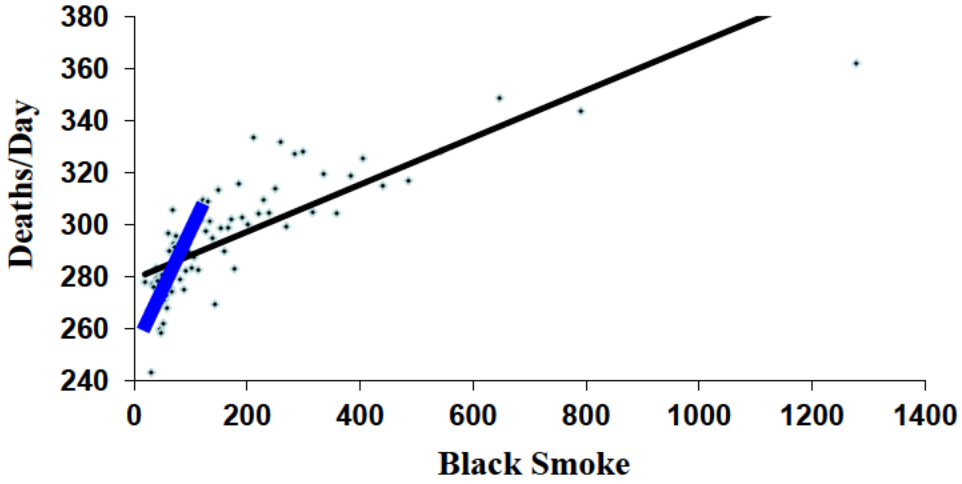


This pattern was not due to influenza, because the epidemic did not hit Britain until about a month later. In the 160 Great Towns of England and Wales (excluding London), the weekly deaths changed from 4,585 in the preceding week to 4,749 in the week of the episode. The increase in pollution was far less in those towns than in London, due to a lack of temperature inversions. Similar associations were reported in other episodes[54]. By their nature, such studies do not tell us what happens at

more typical exposures, but they confirm that particles can kill people rapidly.

In the early 1990's, the results of several daily time-series studies were reported.[32, 55-58] These studies did not rely on extreme pollution episodes, but evaluated changes in daily mortality counts associated with daily changes in air pollution at relatively low, more common levels of pollution. For example, the below figure shows the average deaths in London and black smoke pollution from 14 winters (1958-71), as described by Schwartz and Marcus.[55]

Figure 4. Average deaths and smoke pollution in London, 1958-71



It clearly shows that the association extends to as low as the pollution measurements, and that the slope relating daily deaths to particles is steeper at lower exposure levels. This result has been confirmed multiple times. In particular, large multicity studies of millions of deaths have confirmed that, controlling for season and

weather, when airborne particles increase, daily deaths increase. This has been found in the United States, Europe, and Asia.[59-61] For example, Zanobetti and Schwartz analyzed the association of fine and coarse particulate matter on daily deaths in over 100 cities in the United States and reported strong associations with both.[61]

A recent paper in the New England Journal of Medicine examined the relationship between daily particle concentrations and daily deaths in 652 cities around the world. They reported a significant association with $PM_{2.5}$ after controlling for gaseous air pollutants and temperature, with a larger change in death rate per $\mu g/m^3$ in cities with lower concentrations, such as in the U.S.[62]

Moreover, this is again supported by animal studies and controlled human exposure studies showing that short-term exposure to particles increases blood pressure, decreases arterial diameter, increases inflammation in the arteries, changes electrocardiogram patterns, etc., all of which produce short-term increases in the risk of death from heart disease. Other studies show that particle exposure exacerbates pneumonia in animals, providing support for the studies showing that particles are associated with increased deaths from pneumonia.

Confounding

There is convincing evidence that the association of particles with early death cannot be explained by confounding. The issue here is similar

to that discussed for longer-term exposure above—could some other factor, correlated with both exposure and mortality, explain the observed association. One important difference in these short-term studies of day-to-day changes is that most of the confounders one might worry about in the long-term studies cannot be confounders here. Socio-economic status, body mass index, years of smoking in the population, etc. simply do not vary from day-to-day, and hence cannot possibly confound the association of daily deaths with day-to-day changes in $PM_{2.5}$.

Case-Crossover Studies

The case-crossover design, introduced by Maclure in 1991, is a method for investigating the acute effects of an exposure.[63] In the case-crossover approach, a case-control study is conducted whereby each person who had an event is matched with him/herself at a nearby time period where s/he did not have the event. The subject's characteristics and exposures at the time of the case event are compared with those of control periods in which the event did not occur. If exposure is related to the risk of an event, then we would expect, on average, higher exposure levels on the event days than on the control days. I introduced this approach to studying mortality effects of daily changes in particulate air pollution.

Applied to the association of air pollution with risk of death, the approach has several advantages. Because in this analysis each person serves as his or her own control, the use of a nearby day as the control

period means that all covariates that change slowly over time, such as smoking history, age, obesity, usual diet, diabetes, and so forth, are controlled for by matching. Again this is an approach that makes sure that most predictors of outcome are independent of exposure, producing estimates that can have a causal interpretation. The health profile of the person is the same on the day they died and the nearby control days except for a very few things that can change importantly from day-to-day. Hence, almost all confounding is eliminated by design. The remaining health risks that might have day-to-day fluctuations in parallel with particulate air pollution are other pollutants and weather.

The method also allows a more straightforward approach to seasonal control. Traditional methods involve Poisson regressions with smooth functions or regression splines to control for season. The case-crossover design controls automatically for seasonal variation, time trends, and confounders that vary slowly by time because the case and control periods are separated by a relatively small interval of time. That is, season and time trends are controlled by matching. Bateson and Schwartz demonstrated that by choosing control days close to event days, even very strong confounding of exposure by seasonal patterns could be controlled by design in the case-control approach.[64, 65] This makes the approach an attractive alternative to the Poisson models.

Once one has adopted the framework of choosing control days close to the event day for each subject, it is straightforward to extend this to

control for a gaseous air pollutant. One can examine all of the potential control days that are close enough in time to each event day to control for seasonal confounding, and select the subset that also has the same level of the gaseous co-pollutant as the event day.[66, 67] Since the day of death and control day for each decedent have the same concentration of the other co-pollutant, the association between PM and death (from having on average higher PM on the day of death than the control day) cannot be alternatively explained by the other pollutant, which is the same on both days.

I applied this approach to a study of particulate air pollution and daily deaths in 14 U.S. cities.[67] After matching, there were about 400,000 deaths in each analysis. PM_{10} was a significant predictor of mortality when controlling for gaseous air pollutants, with effect sizes ranging from a 0.45 percent increase in daily deaths per 10 $\mu g/m^3$ increment of PM_{10} [95% confidence interval (CI), 0.12–0.79%] when death and control days were matched ozone levels, to a 0.81 percent increase per 10 $\mu g/m^3$ increment of PM_{10} (95% CI, 0.47–1.16%) when matched on SO_2.

As noted above, the matching of case and control days by SO_2, NO_2, CO, and O_3 unambiguously showed that particles were associated with higher daily death rates in situations where those pollutants could not possibly be alternative explanations of the observed association, because they were the same on the day of death and control day. I did a similar study where the day of death and control day were matched by temperature, and again

showed there was no confounding[66]. Hence, neither temperature nor other air pollutants can explain the observed association.

More recently, we again used our national model predicting $PM_{2.5}$ daily on a 1km grid for the continental U.S.[68] to fit a case-crossover analysis in the entire Medicare population. We looked at 22 million deaths among 61 million Medicare participants in the U.S. and found a highly significant association between $PM_{2.5}$ and daily deaths, which continued well below the EPA ambient air quality standard.

Covariate Adjustment to avoid confounding

Other studies using different methodologies have arrived at the same conclusion. For example, the National Mortality and Morbidity Air Pollution Study (NMMAPS) fit two pollutant models in the 20 largest U.S. cities to estimate whether there were significant associations of particles with daily deaths independent of gaseous pollutants, and found that the association with particles was unchanged by control for gases.[69] In another approach, I used a two-stage hierarchical model to examine confounding by gaseous air pollutants.[70-72] The basic idea of this approach is that the relationship between particles and the putative confounder varies by city and season. If the observed particle effect on daily deaths is really due to the other air pollutant, the size of the estimated particle effect should vary in the same way as that relationship between the particles and the confounder. That is, suppose that the particle

effect was really due to ozone instead. The association of ozone with particles is quite different between summer and winter, and also varies by city. If the observed particle effect were due to ozone, one would expect it to vary between summer and winter, and by city in a manner parallel to how the relationship between particles and ozone varied. It did not in the NMMAPS study, confirming the lack of confounding by other pollutants.

Another possibility considered was confounding by respiratory epidemics. To examine this, Braga, I and other coworkers analyzed daily deaths and PM in five U.S. cities in two ways.[73] First, we did the now-classic Poisson time series analysis. Then, we used hospitalization data for pneumonia to identify each serious respiratory epidemic in each city. We fit 6th degree polynomials for each epidemic in each city to explain its rise and fall with time and compared the association with particles to the original results. There were no changes in the association between daily PM and daily death, with or without control for epidemics.

Another way to ensure another air pollutant does not explain an association is to conduct a study in a location and at a time where that other air pollutant is not present. For example, Fairley examined the association of particles and daily deaths in Santa Clara, California, in the winters.[58] Since ozone is a summertime pollutant, ozone was essentially not present, and since sulfur containing fuels are not used in California, SO_2 levels were essentially non-existent. Yet similar associations with particles were seen as in other cities. Similarly, there is essentially no

SO_2 in Provo, Utah, where particles have been associated with daily deaths.[32] Similarly, the analysis of London winters could not have been confounded by ozone.[55]

The report of Sarnat et al. in Baltimore is also informative on the question of confounding.[74] To argue that the effect of particles on mortality is really due to the fact that particle concentrations are correlated with, for example, SO_2 concentrations, makes an unspoken assumption. It assumes that particle concentrations measured at monitors in a city are related to personal exposure to particles, and that SO_2 concentrations are related to personal exposure to SO_2. Sarnat and coworkers used personal samplers that measured personal exposures to particles and these other pollutants in adults and children in the summer and winter to determine whether this was true. We found that personal exposures to particles and the particle concentrations at monitors were related. They also found that day-to-day variations in gaseous air pollutants measured at the central monitoring stations in the city **were not associated** with day-to-day changes in personal exposures to those gases. However, the monitored values of SO_2 and other gases *were* associated with day-to-day changes in personal exposure to $PM_{2.5}$. Hence, measurements of gaseous pollutants at central monitoring stations are not approximations of exposure to those gases. They are approximations of exposure to *particles*. In contrast, the measurements of particles at the central monitoring stations were correlated with personal exposure to

particles. Hence, ambient measurements of gases **may be alternative surrogates for exposures to particles and not measurements of confounders at all**. This indicates that studies of particles should not control for those gases. Another study in Boston confirmed these results.[75]

Another personal exposure study examined the association among ambient measurements of temperature, personal exposure to temperature, and skin temperature in Baltimore. In this case, as well, ambient temperature was not correlated with either personal exposure to temperature or with skin temperature, indicating little potential for confounding.[76] This is not surprising. Adults in the United States spend over 95 percent of their time indoors, where space heating is ubiquitous and air conditioning common.

Causal Modeling to avoid Confounding

Another causal modeling approach to avoid confounding is called an instrumental variable analysis. The basic idea is that there are many sources of variation in particulate air pollution. If one can identify one of those sources, which is not plausibly associated with any potential confounders, one can use it as a surrogate for exposure, and if it is associated with daily deaths, the association must be causal since it is not confounded. I applied this approach in a study of $PM_{2.5}$ and daily deaths in Boston.[77] I used a model from the National Oceanographic and Atmospheric Administration, which can track back trajectories of where the air over Boston at any given day

was up to 96 hours prior to reaching Boston. I believe these trajectories are a valid instrument since 1) they represent emissions elsewhere, and are unaffected by the behavior of people in Boston; 2) people in Boston are unaware of them, and hence do not modify their behavior based on them; and 3) there is no plausible connection between them and changes in other behavior that influences short-term mortality rates such as number of cigarettes smoked, daily changes in diet, alcohol consumption, etc. The only possible exception is temperature, and so I controlled for temperature. We found a strong association between the instrument (i.e., back trajectories) and daily deaths in Boston.

The above instrumental variable approach identified a causal effect of particles transported from upwind locations. I subsequently fit another instrumental variable model for Boston that focused on particles that were locally generated. The atmosphere close to the ground is characterized by strong vertical mixing, diluting local emissions into the air above and hence lowering the concentrations at ground level. However, the exact height up to which this mixing occurs (the mixing height) varies substantially from day-to-day. On days when the mixing height is higher the same emissions will produce lower concentrations, since they are being diluted into more air, and vice versa. Hence, the mixing height explains some of the variation in exposure to local particles. It is also implausible that it influences the risk of dying through any other mechanism, and hence serves as a good instrument for variations of local particles that are independent of all

confounders. I showed that it, and local particles, were causally associated with day to day changes in how many people died[78]. As an alternative, I used a propensity score model to develop weights that made day-to-day changes in $PM_{2.5}$, *local or transported,* independent of other predictors of death, and showed that this too, produced a significant causal association with daily deaths.

Further, in a recent publication in the official journal of the National Institute for Environmental Health Sciences (part of the National Institutes of Health), I analyzed the relationship between $PM_{2.5}$ and daily deaths in 135 U.S. cities using three different causal modeling methods, and combined the evidence across all the cities[79]. One causal method used daily fluctuations in the height up to which air pollution can get mixed, and diluted, as a surrogate for variations in $PM_{2.5}$ that are due to something that is not itself a cause of death, nor a predictor of day to day changes in smoking, diet, etc. We found a significant association with $PM_{2.5}$. A second method looks at $PM_{2.5}$ exposure after the death occurred as a variable that cannot be causally related to the prior deaths, but could be correlated with the same omitted confounder that was correlated with $PM_{2.5}$ on the day of death. If such a variable exists, then control for $PM_{2.5}$ after the day of death should partially capture its effect and reduce the effect of $PM_{2.5}$ on the day (or day before) the death. This did not happen, indicating that such a variable does not exist. The third approach used the IPW weighting approach, to make $PM_{2.5}$ independent of all the covariates

so it cannot be confounded by them. Again, $PM_{2.5}$ predicted higher death rates.

Averaging Time

Another line of research has sought to bridge the gap between studies looking at annual average (or longer) particle concentrations and annual death rates, and the studies looking at daily particle concentrations and daily deaths. In my own research, I have examined the association of daily deaths[80] and hospital admissions[81] with particles when averaged over different time periods, from days to months, after filtering out seasonal and long-term trends. I found that the size of the PM effect increased as one went from days to periods of up to two months. At that point, the effect size estimates seemed intermediate between those reported in daily time series, which looked at the previous day's exposures, and those reported in the cohort studies, which show larger impacts.

As part of this approach, I developed a framework for thinking about how mortality rates might respond to increases in air pollution. In this framework, the population is divided into a general pool with low risk of dying, and a frail pool with high risk of dying in the near future. Because the causes of frailty can be at least partially reversible (e.g., people with pneumonia recover, people who survive a myocardial infarction have much lower risk of dying in the second month after the infarct than in the first, etc.), it is possible to transition back to the general pool

from the frail pool. Meanwhile, other events can cause transitions from the general pool to the frail pool. If the effect of particles on the recruitment rate into, or retention in, the frail pool is greater than the effects of particle exposure on the death rate out of the frail pool, increased exposure will result in an immediate increase in deaths (from the direct effect of particles on the death rate out of the frail pool). But it will also result in a delayed effect, as the increased population of the frail pool results in excess deaths over the next few months. The results of my studies indicate that this is the case.

A frequency domain regression approach by Zeger and coworkers showed similar results[82]. In several studies, Zanobetti and coworkers examined the time course of the mortality-death relationship directly, using distributed lag models[53, 83]. These models showed a pattern concordant with my hypothesis. There was an immediate increase in deaths following an increase in particle exposure, followed by a long tail of slightly increased deaths, stretching out for 40 days after the initial response. Time series studies by their nature have to control for season, and this makes it difficult to examine lags longer than a month or two, but the substantial increase in effect size reported by Zanobetti in these studies again suggests that the short-term and long-term responses to changes in airborne particles fall on a continuum.

Further support for this theory comes from studies looking at pregnancy outcomes and infant mortality. Both responses, by definition,

involve exposures of less than a year. For example, Bobak and Leon examined the cross-sectional association between air pollution and infant mortality rates across towns in the Czech Republic[84, 85]. A significant association was seen with particle concentrations. Woodruff and coworkers compared infant death rates in U.S. cities with their levels of PM in the air[86]. They excluded infant deaths in the first month after birth as likely to reflect complications of pregnancy and delivery, and found that PM_{10} was associated with higher death rates in the next 11 months of life. This excess risk seemed to be principally from respiratory illness, although sudden infant death syndrome deaths were also elevated. Further studies in later years looking at $PM_{2.5}$ confirmed this association[87, 88].

III.A.2. DOSE RESPONSE AND THRESHOLD

A critical issue regarding PM and health impacts is whether a threshold exists for the effects of particles, and more broadly, what shape the dose-response curve takes. I first summarize several conclusions on this matter by various regulatory and professional bodies, and then discuss my opinion regarding the conclusions reached by these bodies.

After its recent reviews of the scientific literature on particulates and health impacts, EPA concluded that there is no evidence for a threshold. For example, the Regulatory Impact Analysis for the final Transport Rule, published in 2011, states:

> Based on our review of the current body of scientific literature, EPA estimated PM-related mortality without applying an assumed concentration threshold. EPA's Integrated Science Assessment for Particulate Matter (U.S. EPA, 2009b), which was recently reviewed by EPA's Clean Air Scientific Advisory Committee (U.S. EPA-SAB, 2009a; U.S. EPA-SAB, 2009b), concluded that the scientific literature consistently finds that a no-threshold log-linear model most adequately portrays the PM-mortality concentration-response relationship while recognizing potential uncertainty about the exact shape of the concentration-response function[89].

This issue was extensively peer-reviewed by the CASAC, which concurred with the conclusion that there is no evidence for a threshold: "Although there is increasing uncertainty at lower levels, there is no evidence of a threshold (i.e., a level below which there is no risk for adverse health effects)."[8] Similarly, the EPA Policy Assessment, a document that translates the ISA into a focus on policy relevant science and is also reviewed and approved by CASAC, stated: "We note that no discernible thresholds have been identified for any health effects associated with long or short-term $PM_{2.5}$ exposures."

The National Academy of Sciences concurs, stating, "For pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold."[12]

This is also the view of the World Health Organization. The United Nations Environment Program and the World Meteorological Association commissioned a report on the health and climate effects of controlling particles and ozone. As part of this internationally peer-reviewed report, they assessed the mortality effects of reducing particle emissions and ozone precursors. They found no evidence for a threshold in the mortality effects of particles, conducted a risk assessment for the health improvements that would result from the policies they were recommending, and concluded:

> Full implementation of the identified measures could avoid 2.4 million premature deaths (within a range of 0.7-4.6 million) and the loss of 52 million tonnes (within a range of 30-140 million tonnes), 1-4 per cent, of the global production of maize, rice, soybean and wheat each year (Figure 1)[90].

This work has subsequently been published in Science.[91]

Similarly, the Health Effects Subcommittee (HES) of EPA's Advisory Council on Clean Air Compliance Analysis, when asked whether EPA should do risk assessments assuming no threshold, stated that it "*fully supports EPA's use of a no-threshold model to estimate the mortality reductions associated with reduced PM exposure.*"[92]

In the $PM_{2.5}$ NAAQS rulemaking, EPA was asked by the Office of Management and Budget to conduct an expert elicitation on the dose-response relation between particles and deaths and have it reviewed by EPA's external review body, the Science Advisory Board. An expert

elicitation is a well-established process in Decision Science, and in this case involved having an outside contractor with expertise in these elicitations select experts in the field, obtain from them the studies each thought were most relevant to the issue, and make sure all the experts had seen all the studies. The contractor then conducted a structured 8-hour interview with each expert separately. In addition to review by the EPA's external Science Advisory Board, this analysis was published in a peer review journal.

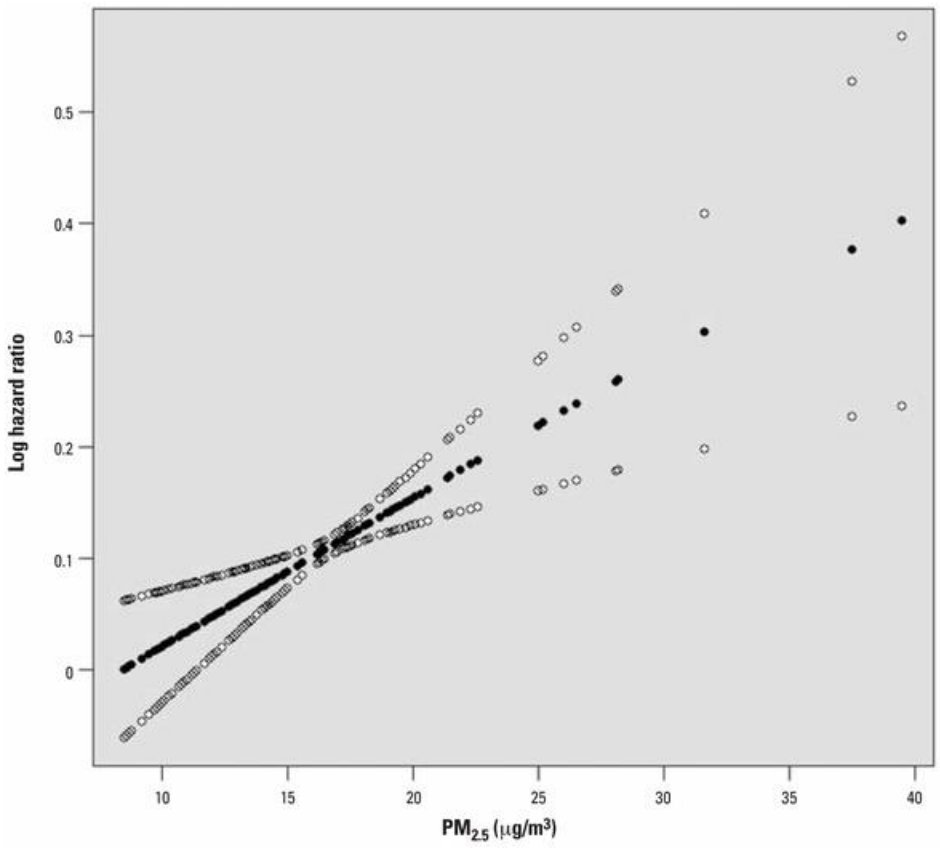
Part of this process addressed the question of a threshold. As noted in EPA's Expert Elicitation Report, 11 out of 12 reviewers believed that there was neither evidence nor even a theoretical basis for a threshold. The remaining reviewer thought there was a 50 percent probability of a threshold, but that if it existed, there was an 80 percent probability that it was below 5 $\mu g/m^3$.

In its most recent statement on air pollution, the World Health Organization stated specifically about $PM_{2.5}$, that "*[s]mall particulate pollution has health impacts even at very low concentrations – indeed no threshold has been identified below which no damage to health is observed*" (http://www.who.int/en/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health).

In summary, EPA, and two external panels of scientific advisors (CASAC and HES), and WHO believe that the effects of $PM_{2.5}$ continue below the NAAQS, with no evidence of a threshold.

It is easy to see why the expert reviewers reached this conclusion. For example, in another follow-up analysis of the Six City Study, I looked at year-to-year changes in particle concentrations to examine two questions: does the dose-response continue below 15 µg/m^3; and what is the lag between change in exposure and change in mortality rate?[93] We used a penalized spline with up to 18 degrees of freedom (essentially, a polynomial with 18 terms to capture any deviation from linearity), and showed that the association was essentially linear down to 8 µg/m^3 where the data becomes sparse, and that the effects of reduced particle exposure on mortality appear to be mostly seen within two years. The figure below shows that association. The dark dots are the dose-response curve, and the open circles represent 95% confidence intervals.

Figure 5. Effects of reduced particle exposure on mortality in a follow-up analysis of the Six City Study

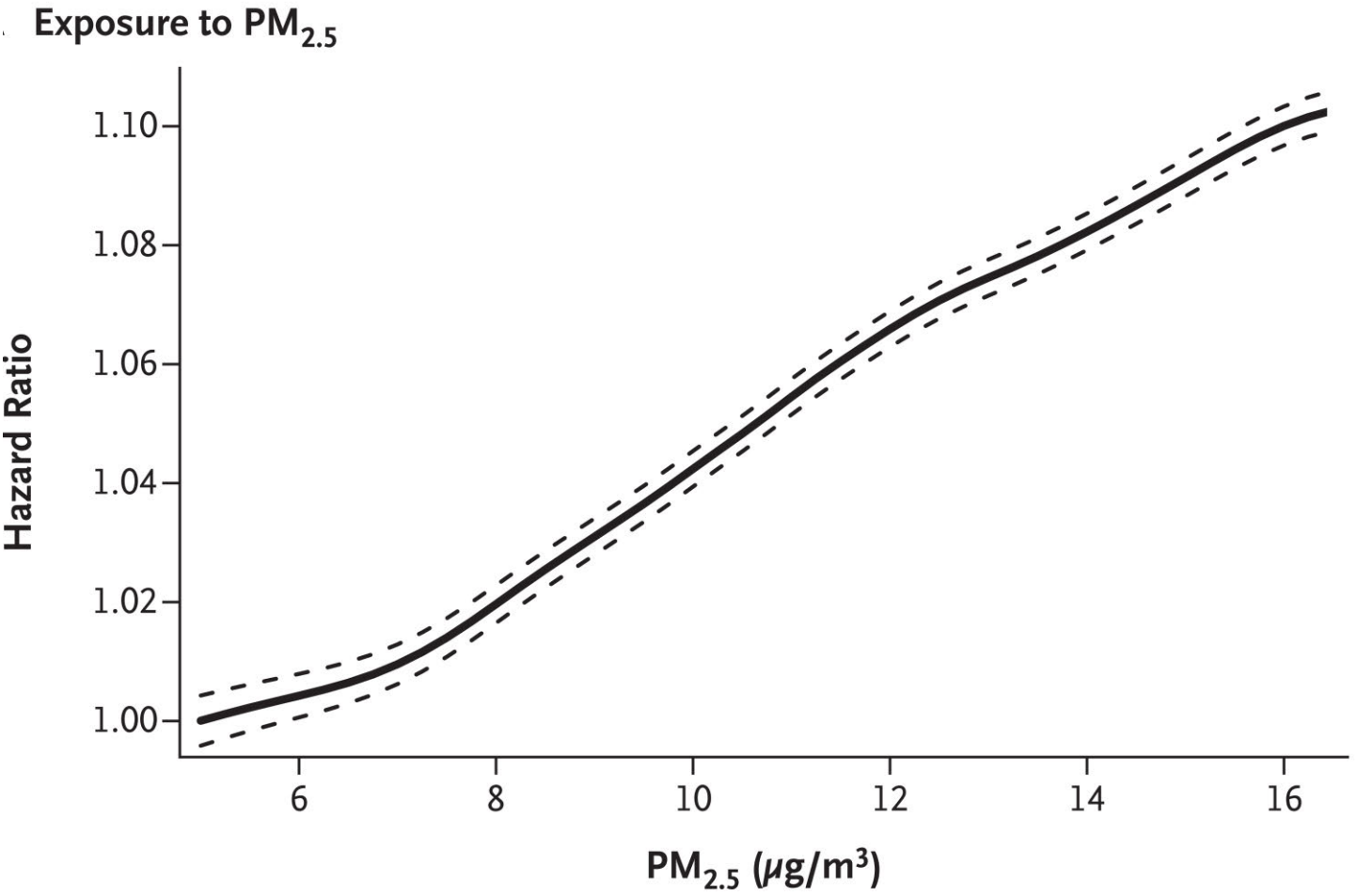


Because the uncertainties around the dose-response curve from fitting a particular model do not reflect the uncertainty in model choice, we also used model averaging, where we explicitly fit 32 models and averaged them, weighted by their probability of being correct given the data.[93] These models explicitly included the possibility of thresholds at multiple different particle concentrations. The association was again indistinguishable from linear with no evidence of a threshold down to the lowest measured level of 8 μg/m^3. Similarly, Pope and coworkers used

nonparametric smoothing to look at the association of $PM_{2.5}$ and mortality in the ACS cohort, and the association was linear from 15 µg/m^3 down to the lowest observed levels (which were also about 8 µg/m^3).[94]

As mentioned above, in Di et al., we examined the association of annual average $PM_{2.5}$ concentrations and mortality rates in 61 million Medicare beneficiaries over 13 years. The extremely large sample size allowed us to estimate the dose-response curve with high confidence. The figure below shows that dose-response curve, with the dashed lines representing the 95% confidence intervals about that line. The Hazard Ratio in the graph is the ratio of the mortality rate at each $PM_{2.5}$ concentration to the mortality rate at the lowest concentration. Hence, the figure indicates that the mortality rate is 2% higher at 8 µg/m^3 than at 6 µg/m^3. The line was estimated as a flexible spline, which would allow it to detect a threshold if one existed. We also explicitly fit a separate model restricted to observations that were below the EPA standard of 12 µg/m^3. When we restricted the analysis to people residing in ZIP codes with annual $PM_{2.5}$ concentration below the 12 µg/m^3 NAAQS, there were 247,682,367 person-years of follow-up and 11,908,888 deaths. We found a 1.36% (95% Confidence Interval (CI) 1.31%, 1.41%) increase in mortality rate per 1 µg/m^3 increase in $PM_{2.5}$ below 12 µg/m^3.

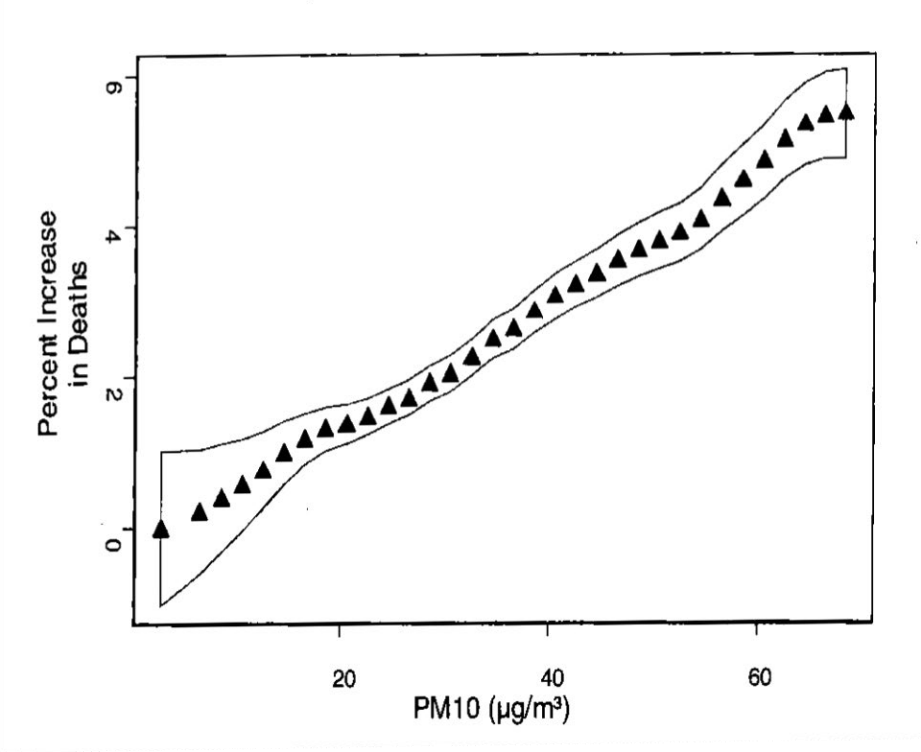
Figure 6. Mortality rates change with $PM_{2.5}$ by ZIP code



A similar result has been seen in studies looking at the acute effect of particle exposure on deaths within a few days of exposure. This has been addressed in a number of large multicity studies. For example, I introduced an approach called meta-smoothing to combine nonlinear dose-response curves across multiple studies.[95] In simulation studies, I showed that it was capable of detecting a threshold, among other forms on nonlinearity, with good accuracy and no bias. This held true even with

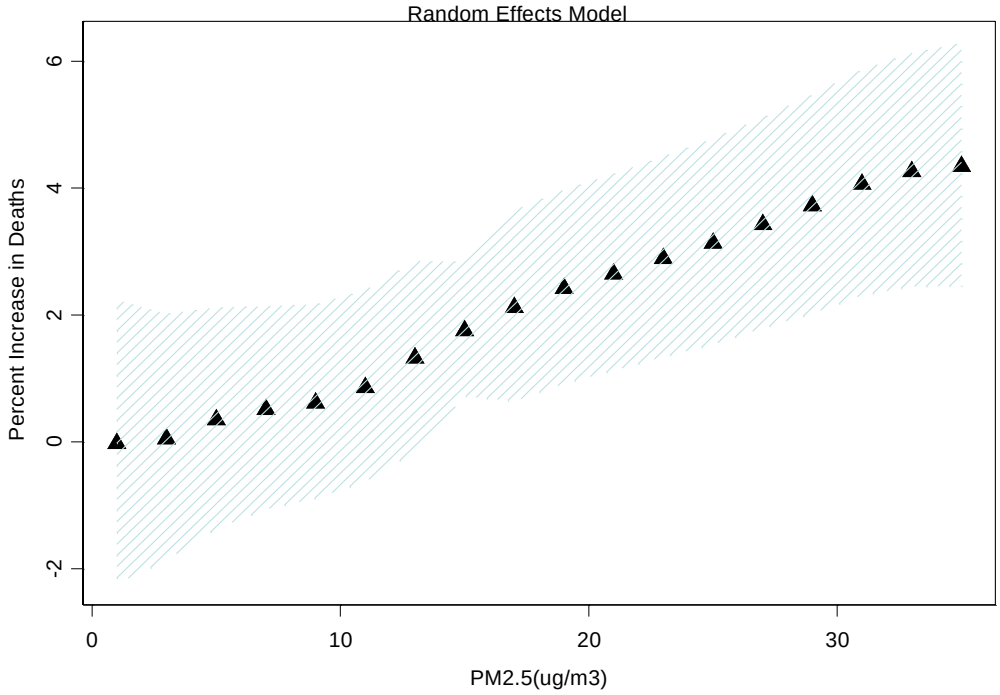
measurement error in exposure. I then applied it to a study of air pollution and daily deaths in 10 U.S. cities. The figure below is the result, showing no evidence of a threshold down to the lowest levels of PM_{10}.

Figure 7. Dose response between PM_{10} and daily deaths in 10 U.S. cities



I also repeated this same analysis using $PM_{2.5}$ as the exposure, instead of PM_{10}, in six U.S. cities where that data was then available.[96] Those results are shown below. Note that essentially the entire dose-response curve takes place on days below the current ambient air quality standard (35 $\mu g/m^3$).

Figure 8. Dose response between $PM_{2.5}$ and daily deaths in six U.S. Cities



Again, there have also been other approaches applied, again with the same conclusion[60, 97, 72, 98, 9972, 98, 99, 71, 96, 70, 95, 69, 94, 68, 93, 67, 92, 66, 91, 90, 65, 89, 63, 87, 88].

Most recently, we published a study of the entire U.S. Medicare population, which suffered over 22 million deaths over a 13 year follow-up[100]. We used a case-crossover analysis and found a significant association of $PM_{2.5}$ on the day of and day before the death with the risk of dying. This association remained significant and the slope increased when the analysis was restricted to days with $PM_{2.5} < 25$ µg/m^3, well below EPA's current 24-hour standard of 35 µg/m^3, and continued well below 10 µg/m3.

The Canadian Community Health Survey cohort, published in 2016, studied 300,000 people across Canada[101]. The mean annual $PM_{2.5}$ concentration in the participants was only 6.3 µg/m^3, a concentration below that prevailing in most of the U.S. Eighty percent of the population was exposed to concentrations below 8.8 µg/m^3, a concentration that was is substantially lower than the NAAQS of 12 µg/m^3. Yet they found a highly significant association of increments in $PM_{2.5}$ across that exposure range with mortality, with a slope of 1.26% increase in mortality rates per 1 µg/m3 increase in $PM_{2.5}$. A formal analysis looking for a threshold found a value of 0 µg/m^3.

As noted by the members of the expert elicitation panel, these empirical results are also consistent with biological understanding. The mechanisms by which particles act include increasing the production of oxidizing chemicals in the body and increasing the amount of inflammation in the lungs, the heart, and systemically. These are not rare processes, where one might think that some effort would be required to overwhelm built-in defenses. Rather, these are processes that are fundamental to the process of aging itself. They are well known to happen at levels above the body's ability to compensate, resulting in decreased lung function over time, increased atherosclerosis over time, etc. Given this, it is not surprising that any incremental increase in these processes produces incremental damage. Hence, one would not expect a threshold to exist.

In addition, the World Health Organization has stated:

"[s]mall particulate pollution has health impacts even at very low concentrations – indeed no threshold has been identified below which no damage to health is observed (http://www.who.int/en/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health)."

III.A.2.a. IMPLICATIONS OF A LINEAR RELATIONSHIP WITH NO THRESHOLD

If, as the data indicate and the scientific community believes, there is a linear dose-response relationship between mortality and $PM_{2.5}$ concentrations with no no-effects threshold, then any increase in particle concentrations in downwind communities of an emission source will result in an increase in the death rate in those communities. While the increased rate will depend on the amount of increase in particle concentrations, and in some cases may be low, it will not be zero. Arguments that the increase in emissions is small and can be considered negligible are therefore, inapt. Small increases in emissions produce small increases in risk, not zero increases in risk. Hence, there will be early deaths and other health effects associated with such an increase over time. This opinion is also supported by the Health Effects Subcommittee (HES) of the Advisory Council on Clean Air Compliance Analysis, another external scientific peer review board for EPA, which as part of a formal review of and EPA analysis of the costs and benefits of the Clean Air Act stated "Further, the HES fully supports EPA's use of a no-threshold model to estimate the mortality reductions associated with reduced PM exposure.[92]"

III.A.2.b. PARTICLE HEALTH EFFECTS AND THE NATIONAL AMBIENT AIR QUALITY STANDARDS

It is sometimes argued that by setting a National Ambient Air Quality Standard for particles, EPA has determined that there are no adverse health effects at concentrations below that standard. EPA has explicitly rejected that interpretation, as noted above. For example, in its Regulatory Impact Analysis for the 1997 $PM_{2.5}$ standard, EPA stated that a NAAQS was not a "no effects" level. Specifically, the agency said "The Act does not require the Administrator to establish a primary NAAQS at a zero-risk level." Subsequently, in its Regulatory Impact Analysis for the Clean Air Interstate Rule (2005), EPA again estimated health impacts of $PM_{2.5}$ using a linear dose-response relationship without a threshold. This is consistent with CASAC approved statements by EPA in its Policy Statement for the $PM_{2.5}$ NAAQS, specifically:

> "We note that no discernible thresholds have been identified for any health effects associated with long or short-term $PM_{2.5}$ exposures." (p ES-1)

EPA has explicitly rejected the view that there are no adverse health impacts from particles at concentrations below the NAAQS and gone further to **explicitly calculate** the lives saved due to further reductions in $PM_{2.5}$ concentrations starting from levels below the NAAQS. It has done this in

the RIA for the $PM_{2.5}$ NAAQS itself. For example, in the RIA for the most recent $PM_{2.5}$ standard, EPA stated:

> "We assume that the health impact function for fine particles is log-linear without a threshold in this analysis. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5}$, including both areas that do not meet the fine particle standard and those areas that are in attainment, down to the lowest modeled concentrations."

And:

> "Therefore, it is not appropriate to estimate the fraction of benefits that occur only in the counties that exceed the standards."

And:

> "The NAAQS are not set at levels that eliminate the risk of air pollution"[102].

EPA's position could not be clearer. As noted previously, EPA concluded in its RIA for the Transport rule that there was no threshold, which was again endorsed by CASAC. Similarly, the EPA Policy Assessment for setting the NAAQS, a document that translates the Integrated Science Assessment (ISA) into a focus on policy-relevant science and is also reviewed and approved by CASAC, stated:

> "We note that no discernible thresholds have been identified for any health effects associated with long or short-term $PM_{2.5}$ exposures."[103]

By definition, no threshold means that any incremental exposure produces an incremental increase in the rate of mortality (or other health effect).

EPA has another external scientific peer review panel that reviews the science and methods used for EPA's estimate of the benefits of the Clean Air Act. The Health Effects Subcommittee (HES) of the Advisory Council on Clean Air Compliance Analysis is charged with reviewing EPA's analysis of the costs and benefits of Clean Air Act regulations. As part of that review, in 2009, EPA explicitly requested comments about a threshold for the effect of $PM_{2.5}$ on mortality, asking

> "Does the Council HES support the use of a no-threshold model for generation of the Primary Estimates of PM mortality incidence reduction?"

The HES responded:

> "The HES fully supports EPA's decision to use a no-threshold model to estimate mortality reductions. This decision is supported by the data, which are quite consistent in showing effects down to the lowest measured levels. Analyses of cohorts using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality."[104]

There is good reason for these conclusions. The Canadian Community Health Survey cohort, published in 2016, studied 300,000 people across Canada[101]. The mean annual $PM_{2.5}$ concentration in the participants was only 6.3 $\mu g/m^3$, a concentration well below that prevailing in most US locations where the

excess NOx emissions occurred. Eighty percent of the population was exposed to concentrations below 8.8 µg/m^3, a concentration that is substantially lower than the NAAQS of 12 µg/m^3. Yet they found a highly significant association of increments in $PM_{2.5}$ across that exposure range with mortality, with a slope of 1.26% increase in mortality rates per 1 µg/m^3 increase in $PM_{2.5}$. A formal analysis looking for a threshold found a value of 0 µg/m^3.

In the Medicare cohort study of Di[27], we followed the entire Medicare population of the United States between 2000 and 2012. When we restricted the analysis to people residing in ZIP Codes with annual $PM_{2.5}$ concentration below the 12 µg/m^3 NAAQS, there were 247,682,367 person-years of follow-up and 11,908,888 deaths. We found a 1.36% (95% Confidence Interval (CI) 1.31%, 1.41%) increase in mortality rate per 1 µg/m^3 increase in $PM_{2.5}$ below 12 µg/m^3.

In conclusion, the setting of a National Ambient Air Quality Standard is not viewed by the EPA as establishing a threshold, and the agency and its scientific advisors, have explicitly rejected that view, and endorsed the lack of a threshold, including in rulemaking documents. Furthermore, the empirical evidence refutes the existence of a threshold, as noted above.

III.A.3. SIZE OF PARTICLES AND HEALTH EFFECTS

Studies on the health effects of particle pollution began years ago, and over time the metric by which airborne particles are measured has

changed. In the late 1980's, EPA measured particles as PM_{10}, particles less than 10 micrometers in diameter. This is small enough to get down peoples' throats and into the lungs. Since the late 1990's, EPA has measured (and regulated) PM_{10} as well as $PM_{2.5}$, which is particles less than 2.5 micrometers in diameter. The $PM_{2.5}$ subset of PM_{10} is thought be more toxic, because combustion particles are all found in that size range, and particles of that size are more likely to deposit deeper in the lung.

A number of studies have documented greater effects of $PM_{2.5}$ on mortality than of coarse particles. Most recently, we examined the association between $PM_{2.5}$, coarse mass (the difference between PM_{10} and $PM_{2.5}$, representing particles with sizes between 2.5 and 10 µM), and daily deaths in 112 U.S. cities. We found each 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 1 percent increase in daily deaths, while coarse mass was associated with about half that increase. The secondary organic and nitrate particles produced by NOx emissions are all in the more toxic, $PM_{2.5}$ range.

III. A.4. PARTICLE COMPOSITION

While the association between exposure to particulate matter mass and mortality is well established, there remains uncertainty as to whether certain chemical components of PM are more harmful to human health than others. To date, the evidence is not convincing that any form of fine combustion particles are more or less toxic than average, with different

studies showing different results. It is important to understand that the conclusion (of the Clean Air Scientific Advisory Committee and others) that we cannot differentiate the toxicity of different types or sources of particles does not mean that we believe it likely that one type of source of particles will ultimately prove to be the "toxic agent." Rather, the consensus scientific opinion is that all airborne particles are toxic, although they may vary in their toxicity. There have been time series studies in locations, such as Santa Clara, California in the winter where wood smoke is the dominant source of particles that show significant associations with daily deaths.[58, 105] There are studies in locations such as Philadelphia where secondary sulfate particles are the major source, which again show day-to-day changes in air pollution are associated with day-to-day changes in deaths.[70, 106-108] In Sao Paolo, Brazil, where traffic particles are the major sources, again, particles are associated with increased deaths.[109, 110] While we have not yet distinguished the relative effects of different sources of particles, it is clear that they all contribute to early deaths.

In the absence of good evidence that any source or type of particle had a *different* impact, the CASAC recommended maintaining a standard for $PM_{2.5}$, that is, treating particles from all sources as having the *same* toxicity. Therefore there is no reason to believe that the organic carbon and nitrate particles formed by NOx emissions have different toxicity than particles in general.

III.A.5 MORBIDITY EFFECTS OF PARTICLE EXPOSURE

A. Long-Term Exposures

Long-term exposure to particulate air pollution is not merely associated with increased risk of dying. It is associated with a wide range of other outcomes, which are in themselves consequential for health. These associations between long-term exposure to particulate matter and morbidity also provide a coherent pattern with the mortality associations.

Lung Function

Lung function is one of these other outcomes that, in my judgment, is clearly affected by particles. Particle levels are associated with less growth of lung function among children and teenagers, and with faster decline of lung function in adults. This is important because lung function is one of the best predictors of cardio-respiratory health and life expectancy.[111, 112] This makes associations between particulate air pollution and lung function relevant for other health outcomes. In the 1950's, a study examined the lung function of British Postal workers, and found that workers in large urban areas with greater pollution consistently had lower lung function. A key advantage of this study was that the postal workers generally stayed in the same counties all of their careers and were of the same social status. Since then, many other studies

have reported associations between lung function and particles in the communities where people resided.[113-118]

Another example is the paper of Lubinski and coworkers, who reported decreases in the ratio of forced expiratory volume in one second (FEV1) to forced vital capacity (FVC), and in FEV1 as a percent of predicted, associated with increased exposure to particles in nonsmoking young Polish men.[119] While these studies looked at differences in PM exposure **between** communities, a study of the rate of lung function growth in children, using data from **within** city variations in Mexico City, reported reduced rates of growth associated with living in parts of the city with higher particle levels.[120] The Southern California Children's Cohort collected extensive information on individual factors that may affect respiratory health, followed the children for eight years and found that $PM_{2.5}$ exposure where they lived was associated with clinically impaired lung function at 18 years of age.[115] By this age the lung has stopped growing for females and almost stopped for males, so these impairments are likely permanent.

Again, a study that looks at differences in exposure over **time**, instead of over **space**, cannot be confounded by variables that differ across location. Such a study would provide strong support for the causality of the associations reported above. A dramatic example of such a study looking at change in exposure was seen by Avol and coworkers.[121] They identified 110 children from the Southern California Children's Health Cohort who moved from the study area, and followed them in their new home

with pulmonary function testing identical to that in the main cohort. Subjects who moved to locations with higher PM_{10} concentrations had lower rates of annual growth in lung function, while children who moved to locations with lower PM_{10} levels than they had left showed higher rates of growth in lung function.

A similar finding has been reported in adults. The SAPALDIA study discussed above is a prospective study of a random sample of adults in eight Swiss communities who were given lung function tests in 1991 and again in 2002.[33] The initial study reported lower lung function in communities with higher particles, controlling for individual risk factors.[118] The follow-up used sophisticated air dispersion models to estimate exposure at each person's address, accounting for any changes in address over the intervening 11 years. In analyses adjusted for town of origin, covariates, and baseline exposure, a decrease of 10 $\mu g/m^3$ in the average subject specific PM_{10} concentration between examinations was associated with a 9 percent decrease in the annual rate of decline in FEV_1 (forced expiratory volume in 1 second) (i.e., by 3.1 ml/yr; 95% CI, 0.03 to 6.2) and a 16 percent decrease in the annual rate of decline in forced expiratory flow (FEF) between 25 percent and 75 percent of volume out (FEF_{25-75}) (i.e., by 11.3 ml per second/yr; 95% CI, 4.3 to 18.2). This annual decrease in lung function is a strong predictor of survival—people whose lung function declines more rapidly as they age have a shorter life expectancy. Hence, these results are consistent with the finding that

decreasing exposure results in increased life expectancy, reported earlier.

Another study took advantage of a natural experiment: the collapse of communism. Sugiri and coworkers examined lung function in East and West German children repeatedly between 1991 and 2000.[122] In 1991, the West German children had higher lung function and much lower exposure to particles. By 2000, changes in the East German economy had eliminated the difference in particle exposure, and the difference in lung function also disappeared.

Recently, Lepeule and colleagues looked at exposure to traffic particles and lung function decline in an elderly cohort in Boston. Participants with higher particle exposure had a more rapid decrease in lung function over time[123]. Again, these findings support the association of particle exposure with greater death rates, since lung function is one of the strongest predictors of life expectancy.

These studies are supported by toxicology, as discussed in more detail below. For example, one study exposed mice and their offspring to either ambient air, or air filtered to remove only particles, not gases. Lung function in the particle-exposed mice, with average particles concentrations of 16.8 $\mu g/m^3$, was lower than in the mice with filtered air, with particle concentrations of 2.9 $\mu g/m^3$.[124]

Respiratory Symptoms

I also find convincing evidence that long-term exposure to particles is associated with increases in bronchitic symptoms, chronic cough, and other serious respiratory illnesses.

Dockery and coworkers, comparing symptom reports across six communities in the eastern United States with varying levels of pollution, and controlling for individual risk factors, found that chronic bronchitis and chest illnesses in children were associated with exposure to particulate air pollution.[125]

Subsequent studies in the United States and Europe confirmed that particle exposure across communities was associated with higher rates of chronic cough and bronchitis symptoms in children, but not with wheezing and asthma. For example, a large study (n=4,470) examined school children in 10 communities in Switzerland and reported an odds ratio for bronchitis of 2.88 (95% CI 1.69–4.89) for PM_{10} exposure between the most and least polluted community.[126] That is, rates of bronchitis were almost three times as large in the highest PM community. The previously mentioned Southern California study examined 3,676 children across 12 communities and found that bronchitis was associated with PM_{10}, but only among children with asthma.[127] The largest study was the Harvard 24-city study, which examined 13,369 children. Particulate air pollution was associated with bronchitis episodes across these communities, controlling for individual risk factors.[128]

Again, these studies have been complemented by studies examining **change** in pollution and **change** in symptom status. A follow-up of the Swiss children's study mentioned above reported that *decreases* in PM_{10} were associated with *decreased* respiratory symptom reporting in the 10 communities.[129] Another follow-up of the SAPALDIA study reported that changes in PM_{10} exposure estimated at adults' addresses were associated with changes in chronic respiratory symptoms during the 11-year follow-up.[130] Similarly, declines in particle levels in East German towns following the collapse of communism were associated with declines in the rate of bronchitis and chronic cough in children.[131]

Lending further credence to these reports is the study of Giroux and coworkers that contrasted exhaled NO (a marker of lung inflammation) in asthmatic children living in urban areas with others staying in a national park in the mountains.[132] They found that the exhaled NO concentrations in the urban asthmatic children were more than double those in the asthmatic children in the national park, indicating that urban air pollution is associated with pulmonary inflammation.

Long-term exposure to PM also has been associated with the development of chronic respiratory symptoms in adults. For example, I reported in 1993 that chronic pulmonary symptoms such as bronchitis were associated with long-term exposure to air pollution in the National Health and Nutrition Examination Study (NHANES) II study adults.[133] Using data from the Seventh Day Adventist Study, a number of papers have reported

associations between particle exposure and chronic respiratory symptoms, most recently using $PM_{2.5}$ as the exposure.[134] Avino and coworkers also reported an association in a more limited two-community study.[135] An interesting case-control study by Karakatsani used in-home examinations by physicians, including pulmonary function testing, to confirm self-reports of chronic respiratory conditions in adults, and in age and gender-matched controls. A geographic model was used to assign individual exposure values, which were associated with the risk of chronic bronchitis and chronic obstructive pulmonary disease (COPD).[136]

Heart Disease

Studies have also linked long term exposure to particles with the development of heart disease, including atherosclerosis, increased blood pressure, as well as more heart attacks and strokes. For example, Kloog matched the entire Medicare population of New England to $PM_{2.5}$ estimates at their ZIP code, and showed that independent of the effects of short term exposure to particles on the risk of heart attacks and strokes (i.e. as a triggering mechanism) long term exposure was associated with substantially larger estimated increases in heart attack and stroke rates[137]. A follow-up of over 6500 patients in Ohio undergoing cardiac examinations found that $PM_{2.5}$ was associated with increased risk of severe atherosclerosis and of having a myocardial infarction (heart attack) in the next three years.[138] Another study followed up post MI patients to see which ones developed

clinical frailty over 10-13 years of follow-up. $PM_{2.5}$ exposure increased that risk[139]. Another twenty-year follow-up study showed that $PM_{2.5}$ exposure increased the risk of death in post MI patients, and that increase was larger in frailer people[140].

Cognitive/neurotoxic effects

There is now abundant toxicological and epidemiologic evidence showing that exposure to particulate matter at commonly occurring concentrations in the air results in acute and chronic inflammation, including in the brain. For example, Calderón-Garcidueñas and coworkers have shown in a series of studies that Mexico City air pollution was associated with central nervous system (CNS) inflammatory and neurodegenerative changes in humans and animals, with increased brain beta amyloid proteins (the proteins linked to Alzheimer's disease) in humans, etc. Specifically, the dogs from Mexico City had greater rates of prefrontal lesions, neuro-inflammation, gliosis, and particle deposition inside the brain. Importantly, brain levels of amyloid-β42, a pathologic hallmark of Alzheimer's disease, were also higher among residents of the polluted cities.[141]

While this study involved air pollution in general, in a follow-up study, Guerra et al. exposed rats to Mexico City particles for two months and reported upregulation of inflammatory genes and elevated levels of inflammatory proteins in the brains of the exposed animals.[142] Fonken and

coworkers exposed mice to either filtered air or $PM_{2.5}$ for eight months and found particles produced increased inflammation in the hippocampal area of the brain, which is central to learning and memory.[143] Further, the particle-exposed animals showed increased depressive symptoms and decreased spatial learning and memory.[143] The table below summarizes recent toxicological studies of neurotoxicity. A major finding of these studies was increased brain inflammation (NF-κB, TNF-α, and IL-1α are inflammatory proteins).

Table 1. Toxicological studies of PM and neurotoxicity

Study	Findings
Campbell et al. (2005)[144]	Two-week near-highway PM exposure in ovalbumin sensitized mice led to increased brain NF-κB and IL-1α
Cruits (2008)	Diesel exhaust exposure caused an increase in fast-wave EEG activity in humans
Veronesi et al. (2005)[145]	Five-month exposure to ultrafine/fine PM-induced loss of dopaminergic neurons in substrantia nigra of ApoE -/- mice
Kleinman et al. (2008)[146]	Six-week exposure to concentrated near-highway PM in ApoE null mice led to activation of inflammatory mediator

	transcription factors
Gerlofs-Nijland et al. (2010)[147]	Four-week Diesel exhaust exposure induced elevations in rat striatal TNF-α and IL-1α
Suzuki et al. (2010)[147, 148]	Mice exposed in utero to Diesel PM had reduced locomotor activity
Fonken et al. (2011)[143]	Ten-month $PM_{2.5}$ exposure led to oxidative stress and inflammatory changes in mouse hippocampus and to decreased learning and memory
Allen et al. (2013)[142, 149]	Mice exposed as neonates or adults to ultrafine PM have preference for immediate reward upon behavioral testing
Guerra et al. (2013) [142]	Two-month Mexico City ambient PM exposures in rats led to region and PM size-specific increases in oxidative stress, inflammation, and unfolded protein responses

Human Studies of Particles and cognitive function

Since 2008, there has been a rapid expansion of human studies on neurological effects of particles. Because the brain is functionally

rewiring and developing in early childhood, and because cognitive function begins to decline after 50, children and the elderly have been the focus of these efforts. Franco-Suglia et al. compared cognitive function of children to their lifetime particle exposure.[150] We used a geographic exposure model, calibrated to 82 different monitoring locations, to estimate the geographic variation of traffic particles in Boston. From this model, we estimated chronic exposure to traffic particles for school-aged children living in Boston. Higher particle exposure was associated with decrements in cognitive function of the order of two IQ points.

Another study of 671 older men living in the Boston area again used exposure to black carbon, a marker of traffic-related particles, and observed that higher level of exposure over the previous one to 11 years was associated with worse cognitive function.[151]

In a study of 15,973 older adults in China, residents of areas with poorer air quality over the previous seven to 10 years, measured by an index of ambient particulate and gas concentrations, were more likely to have poor cognitive function.[152] More recently, an even larger study of 19,409 U.S. nurses developed land-use regression exposure models that predicted long-term $PM_{2.5}$ and PM_{10} concentrations at the addresses of each nurse. Nurses with higher baseline exposure (that is, before the first cognitive exam) had faster rates of decline in cognitive function over time.[153] The Project Mobilize study, reported an association with residential proximity to major roadways, but not with black carbon, and

impaired cognitive function in elderly inhabitants of Boston.[154]

In another study of older subjects, Ranft and coworkers found associations between long-term particle exposure and cognitive function in elderly women,[155] and Chen and Schwartz reported associations with ozone and, more weakly, with PM_{10} in the NHANES III study.[156]

More studies of children have also appeared. In 2009, Freire and coworkers reported an association of traffic-related air pollution and cognitive function in a large, well-controlled cohort study in Spain.[157] Simultaneously, Wang et al. published a study comparing cognitive function in schoolchildren in a high and low-traffic neighborhood in Quanzhou, China.[158] The children from the higher-traffic school had lower cognitive performance. More recently, Gatto et al.[159] and van Kempen et al.[160] have also reported associations, particularly with traffic pollutants, with cognitive performance in children. Traffic is a major source of particles in the air.

Second-hand tobacco smoke particles have similarities with ambient particles, as second-hand smoke is enriched in combustion particles compared to primary tobacco exhaust, which has more gases. Consistent with this, Rauh et al. measured prenatal and post-natal exposure to environmental tobacco smoke, or ETS, and Bayley Scales of infant development in 226 urban children enrolled during pregnancy and followed longitudinally.[161] Prenatal ETS exposure (dichotomized as yes/no) predicted a 5-point decrement in the Bayley MDI scores ($p=0.02$).

Autism and Other Neurological Diseases

Cognitive function is not the only neurological function that has been linked to particle exposure. There is growing literature associating air pollution, and especially particles, with increased risk of autism. A case-control study in Los Angeles reported an association between residence close to a freeway during pregnancy and the risk of being diagnosed with autism.[162] A follow-up study reported that an 8.7 $\mu g/m^3$ increase in $PM_{2.5}$ exposure during pregnancy was associated with more than a doubling of the odds of having an autistic child.[163] Becerra et al. also reported an increased risk of autism with increased particle exposure during pregnancy.[164] Using prospective cohort data from the Nurses' Health Study II Cohort, researchers compared the exposure to air pollution during pregnancy of participants' whose children developed autism spectrum disorder (cases) with that of controls without autism spectrum disorder, and found that fetal exposure to $PM_{2.5}$ at the home address was associated with increased risk of autism spectrum disorder[165].

In addition, Kioumourtzoglou demonstrated, again using a differences in differences causal approach, that long-term exposure to particles was associated with higher rates of hospital admissions for neurological conditions, including dementia and Alzheimer's disease.[166] And Mehta showed effects of particle exposure on psychosocial stress in an elderly cohort.[167]

Relapses of multiple sclerosis are also related to neurological inflammation and hence plausibly to particle exposure. Recently a case-crossover study of a cohort of MS patients showed that particles were significantly associated with relapses of severe symptoms[168]. Another study in Italy found an association of particles with hospital admissions for MS relapses[169].

We have also seen two studies examining the relation between particle exposure and measurements of the brain using magnetic resonance imaging (MRI). Wilker used MRIs from the Framingham Heart Cohort and estimated long-term exposure to $PM_{2.5}$ at subjects' home address using satellite remote sensing data.[170] She found a 2-$\mu g/m^3$ increase in $PM_{2.5}$ was associated with -0.32 percent smaller total cerebral brain volume. Simultaneously, Chen used MRIs from the Women's Health Initiative and reported PM associated with lower white matter volume. Consistent with this, a follow-up study by Calderon-Garciduenas demonstrated increased white matter damage in the brains of dogs living in Mexico City compared to a control community, as well as leaking capillaries and extravascular lipids in the white matter of children in Mexico City compared to the control communities[171].

Another key discovery is that particles have been shown in experimental protocols to translocate from the nose, up the olfactory nerve, and into the brain, with exposures shown not only in the olfactory bulb, but also in the striatum frontal cortex and cerebellum.[172-174]

Overall, I believe this body of research demonstrates that particle exposure is associated with reduced cognitive function in children, as well as lower function and greater cognitive decline in adults. These decreases have important implications. In a meta-analysis of studies relating cognitive function of children or young adults to earnings and labor force participation, I found that a one IQ point decrease in ability was associated with a 1 percent reduction in lifetime earnings, lower probability of attending college, and more time spent unemployed.[175] Hence, the reduced cognitive ability due to particle exposure has an important consequence. Similarly, faster rates of cognitive decline in the elderly can result in substantial increases in cost of care. Decreased cognitive function in the elderly is associated with faster onset of dementia, including Alzheimer's disease. Together with the evidence of increased beta-amyloid protein in the brains of particle exposed humans and animals, this suggests that some of the burden of Alzheimer's disease is due to $PM_{2.5}$ as well. To see the importance of this, one paper has forecasted that a broadly applied intervention that delays the onset of Alzheimer disease by two years could reduce the number of prevalent cases in the United States by about 2 million over a 40-year interval.[176] Finally, the associations with autism and multiple sclerosis, while still early, are plausible given the demonstration that particles induce brain inflammation. Given the devastating nature of these diseases, this

suggests reasonable precaution would dictate reducing exposure whenever it is possible.

B. Short-Term Exposures

Short-term exposures to $PM_{2.5}$ are also linked to a wide range of health effects, including triggering heart attacks, exacerbating respiratory infections to the point where they require hospitalization, triggering asthma attacks, etc. In the section below, I briefly describe the evidence for some of these effects. In particular, hospital admissions for heart disease and lung disease increase following increases in particles in the air. This has been demonstrated in numerous studies over the last several decades, as set forth below.

Respiratory Disease

Bates and Sizto first reported that particles were associated with hospital admissions for respiratory disease in Ontario back in the 1980's.[177] Since that time, numerous studies have reported associations of particles and respiratory admissions in Europe,[178-180] the United States,[181-185] and Canada.[186] These were followed by larger, multicity studies that provided more stable estimates and made use of the same approaches described for the time series mortality studies to assure that associations were not confounded by other pollutants and temperature.[181,

[182, 187-190] In addition, several single city studies took advantage of special circumstances to rule out confounding.

For example, I found that particles were associated with respiratory admissions in Spokane, Washington, a town with essentially no SO_2, and where there was no correlation between particles and temperature, consequently ruling those two out as potential confounders.[190] I also showed that excluding very hot and very cold days, when one would expect the greatest effect of temperature, had no impact on the particle effects. Other studies have confirmed this association in Italy[191]. Again, in a study of the entire Medicare population of the Mid-Atlantic States, we found an association between $PM_{2.5}$ in their ZIP code of residence and hospital admissions for respiratory (and cardiovascular) disease.[192] In addition, a study of seven birth cohorts in Europe found that particles were associated with an increased incidence of pneumonia in infants[193].

Again, this is consistent with animal experiments, where, for example, rodents infected with streptococcus and then exposed to particles had double the bacterial burden and extent of pneumonia as infected animals exposed to filtered air.[194]

Causal Modeling of Respiratory Effects

Kim et al. analyzed the natural experiment of the 1997 fires in Indonesia. Wind patterns swept the smoke across Indonesia with different locations getting more or less exposure depending on details of the

weather patterns. This exposure should be independent of any predictors of health in the population, and hence provide a causal estimate of the effects of a short-to-medium term exposure. They compared the health of people who were in both the 1997 and 2007 Indonesian Family Life Survey, a longitudinal survey of a sample of household across Indonesia. The surveys measured lung function and collected questionnaire data on various measures of health. They estimated exposure using satellite remote sensing measurements of the smoke plume. To further assure the results were causal they used the 1997 results (for lung function, symptoms, health status etc.) as a control for baseline health, socioeconomic status and other possible confounders, and added individual level measures (age, socioeconomic status, etc.) in addition. Controlling for these, they examined the impact of the exposure to smoke from the fires in 1997 on health 10 years later. They found that lung function and a general measure of aggregate health were worse 10 years after the exposure.

Heart Disease Hospital Admissions

Hospital admissions for heart disease are also increased when particle concentrations go up. This is seen for all heart disease admissions, as well as for important categories of cardiovascular disease, such as heart attack, heart failure admissions, arrhythmia admissions, and strokes. For example, in a 1995 study in Detroit, I reported that heart attacks, as well as admissions for heart failure, increased as PM_{10} rose.[195]

Again, when I looked at a city where PM_{10} was essentially uncorrelated with SO_2 or ozone, I found the same result.[196] Studies such as these have again been followed by large multicity studies reporting associations with particles that are not confounded by other pollutants or weather.[182, 189, 197]

Heart Attacks

The association of particles with an immediate increase in hospital admissions for heart attacks indicates that particles must be triggering the occurrence of heart attacks. There is considerable other evidence to suggest that is the case. First, studies have interviewed patients who survived heart attacks and, using case-crossover analyses, found associations with particles.[198] Other focused studies had mixed results.[199-201] However, a large study of 21 U.S. cities definitively confirmed the association.[202] A recent study examined emergency visits for acute coronary syndrome, essentially an early stage of a heart attack, and confirmed an association with particles.[203]

Associations are also seen with long-term exposure to particles and the rate of heart attacks. For example, Madrigano examined participants in the Worcester Heart Attack Study, one of only two myocardial infarction registries in the U.S. She found that $PM_{2.5}$ exposure at residential address was associated with an increased risk of heart attacks[204].

Among approximately 6,600 people undergoing diagnostic evaluation for cardiovascular disease in Ohio, $PM_{2.5}$ exposure was associated with both

increased risk of severe atherosclerosis, and risk of having a heart attack in a three-year follow-up.[138] Similar results are seen in other countries.

The ESCAPE study in Europe followed over 100,000 people who were free of coronary events at intake for 11.5 years, and found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 26% increase in the risk of a heart attack on follow-up[205]

Moreover, there is considerable toxicological and mechanistic support for this effect. This includes experimental studies showing that particle exposure decreases the stability of atherosclerotic plaque.[206] These are discussed below, under mechanistic studies.

Heart Failure

Heart failure is a serious disease where the heart's ability to pump blood to the body is impaired. Patients with heart failure have significantly reduced life expectancy, and episodes of increased impairment are dangerous and frequently result in hospitalization. My study in 1995 identified increases in heart failure after increases in PM_{10} concentrations, indicating that particle exposure could cause an imbalance of the compensation that the body (with medication) makes for the poor pumping ability of the heart.[195] Since then, those associations have been confirmed, including in large multicity studies.[182]

Stroke

The finding that particles increase strokes, a severely debilitating disease, was first reported in Asia.

Recently, this has been confirmed in large multicity studies in the United States.[1,183182, 207-209, 207-209, 207-209, 208-214,198-200] Interestingly, the association seems limited to ischemic strokes, not hemorrhagic strokes, which is consistent with the association between particles and heart attacks.

Diabetes

A prospective cohort study in Los Angeles followed women without diabetes for 10 years and reported associations of new onset type 2 diabetes with both $PM_{2.5}$ and NO_2.[210] An analysis of 23-years of follow-up of participants in the Nurses' Health Study and the Health Professionals Follow-Up Study found that living within 50m of a major road, compared with those living >200m away, was associated with new-onset type 2 diabetes[211]. In addition, a recent meta-analysis by Eze et al., which included three studies on $PM_{2.5}$ and four studies on NO_2, showed an 8–10% increased risk of type 2 diabetes per 10 $\mu g/m^3$ increase in exposure to both pollutants[212]. Support for these finding comes from other studies showing associations between $PM_{2.5}$ exposure and increased serum glucose or impaired glucose tolerance, known risk factors for diabetes[213-215]. Consistent with this, animals exposed to $PM_{2.5}$ demonstrated increased insulin resistance

compared to control animals[216]. This is consistent with a general association of $PM_{2.5}$ with diseases of aging.

III.A.6. MECHANISTIC STUDIES OF PARTICLES

Introduction

The principle diseases caused or exacerbated by $PM_{2.5}$ are cardiovascular, pulmonary, or neurological, so it is important to identify mechanisms by which $PM_{2.5}$ is affecting those systems.

In terms of coronary heart disease and other circulatory diseases, atherosclerosis is a major cause; atherosclerosis is when arteries become clogged with collections of fatty substances referred to as plaques or atheroma. Plaques can cause arteries to narrow and harden, restricting blood flow. Rupture of a plaque can cause a blood clot which subsequently blocks a downstream blood vessel, interrupting blood flow to tissue, and leading to a heart attack or stroke if the blockage is in the heart or brain, respectively. Evidence cited below demonstrates that exposure to $PM_{2.5}$ increases the likelihood that these plaques rupture, by generating an inflammatory response in the lung and release of inflammatory mediators into the arterial circulation, adversely affecting the cardiovascular system – for example, by increasing circulating platelet stickiness, inflammation in the arteries, or oxidative stress (which describes the presence of an unusual concentration of oxidizing compounds). When plaque does rupture, the piece that breaks off can block blood flow in downstream

smaller arteries, cutting off blood supply to part of the heart or brain. Other studies have shown that $PM_{2.5}$ exposure worsens this process as well.

Lung disease is primarily caused by inflammation and oxidative stress in the lung, and studies have indicated that $PM_{2.5}$ causes that as well. In addition, irritation in the lung can trigger nerves, called C fibers, whose activation feeds back into the electrical control of the heart.

In terms of the central nervous system, most neurological diseases result from either inflammation or oxidative stress in the brain or from occluded arteries, small strokes, or severe strokes causing brain tissue to die. Again, the mechanistic studies highlighted below demonstrate that such events do occur, making the epidemiological studies biologically plausible.

Oxidative Stress

Animal experiments indicate that reactive oxygen species (such as hydrogen peroxide, superoxide, etc.), which have established relevance in the pathogenesis of cardiovascular disease and aging,[217] are affected by particles,[218-221] which represent one pathway for their cardiovascular and lung effects. For example, Figure 9 below shows the increase in reactive oxygen species in animals within hours of exposure to concentrated particles from Boston air. Even more impressive, when animals that had been breathing Boston air (without concentration) were placed in a chamber where they could breathe filtered air, the concentrations of reactive

oxygen species in the heart and lung fell by a third within days of removing the exposure (Figure 10).[222] Moreover, it is evident from the figures that levels were still falling when the study was terminated.

Figure 9. Increases in reactive oxygen species concentrations with PM exposure

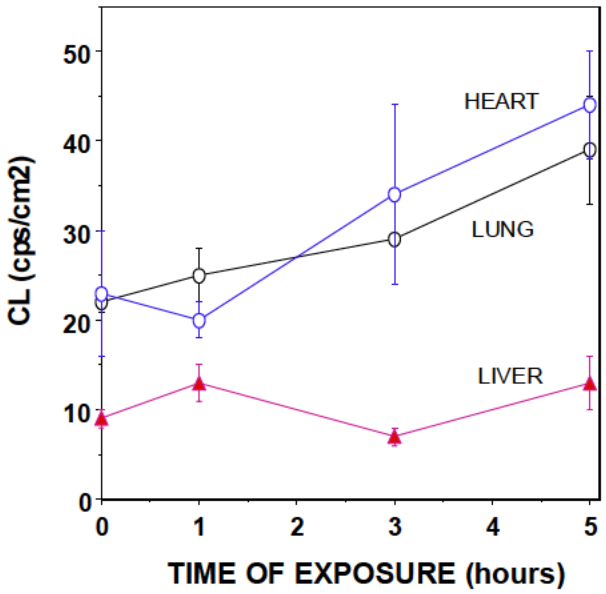
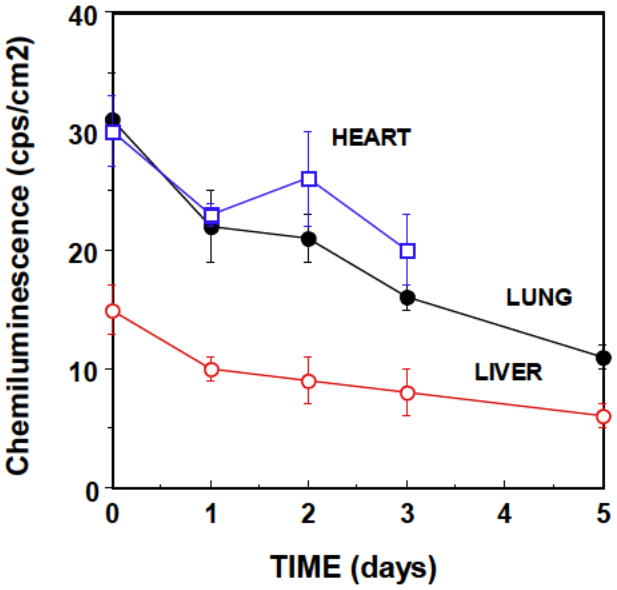


Figure 10. Decreases in reactive oxygen species following removal of PM



A series of studies has shown black soot particles from diesel exhaust to increase oxidative stress in endothelial tissue,[223, 224] inducing the production of heme oxygenase-1, a rapid-response part of the bodies' defense system against oxidative stress. The viability of cell cultures of microvascular endothelial cells was impaired by diesel particles with an accompanying large increase in induction of heme oxygenase-1.[224]

Other studies have examined the role of genes related to oxidative stress on cardiovascular effects of particles, particularly on heart rate variability (HRV). Schwartz[225] and Chahine[226] have reported that subjects missing the gene that makes glutathione S Transferase M1 (part of the defense against systemic oxidative stress), or with a variant of the *HMOX-1* gene that is less good at producing the antioxidant heme oxygenase, had larger effects of particles on HRV, including a significant three-way interaction. Cujuric and coworkers showed the same genes modified the PM-associated decline in lung function.[444] Madrigano showed that one of those genes modified the effects of particles on markers of inflammation in arteries.[227] Ren found that oxidative stress genes modified the PM-induced increase in homocysteine (which increases atherosclerosis).[228] Other studies have focused on oxidative stress per se. For example, Rossner and colleagues examined bus drivers in Prague, and reported increased levels of indicators of oxidative stress such as F-2 isoprostane and 8-hydroxy-

2´-deoxyguanosine (8-OHdG) in drivers compared to controls.[229] Romieu measured malondialdehyde, another marker of oxidative stress, in children and found it associated with $PM_{2.5}$.[230] Ren found that 8-OHdG, a marker of oxidative damage to DNA, was increased by particle exposure, and that the increase was modified by oxidative stress genes.[231]

Inflammation

Particles in the air have been shown to induce increases in inflammation in the lung, as well as systemically (which affects the heart). For example, exhaled NO is a marker for inflammation in the lung. Several studies have shown that particles increase exhaled NO in both children and the elderly.[232, 233] The urinary excretion of 8-OHdG often has been used as a biomarker to assess the extent of repair of DNA damage (induced by inflammation and oxidative stress) in both the clinical and occupational setting. Particles, and particularly the metals on particles, have also been associated with an increased production of 8-OHdG.[231, 234-239] 8-OHdG was also elevated in urban children compared to rural children.[240]

CRP, ICAM-1, VCAM-1, and homocysteine are blood markers of the interrelated processes of inflammation and endothelial function which play important roles in heart disease and atherosclerosis.[241, 242] Accordingly, CRP, ICAM-1, VCAM-1, and homocysteine have been shown to be independently and jointly associated with increased cardiac risk.[243-248] In a prospective study of 28,263 healthy, post-menopausal women, for example, increased CRP

and ICAM-1 were associated with increased risk of cardiac events. Correspondingly, elevated levels of ICAM-1 were associated with the development of accelerated atherosclerosis in a case-control study of 14,916 middle-aged men,[247] while VCAM-1 predicted hospital events in angina patients.[247] Homocysteine inhibits NO release.[248] NO is the principal antioxidant in the artery, and stimulates the relaxation of the muscles surrounding the artery. It has also been associated with coronary artery disease measured radiographically[249] and with flow mediated dilation (the ability of the artery to expand in response to demand for more blood, which is impaired by atherosclerosis).[250] Hence, examining these markers in response to air pollution is important.

Particles have been shown to increase sICAM-1 and sVCAM-1 in diabetics,[251] a finding confirmed in a controlled human exposure chamber study.[218] Particle associations with CRP have been mixed in studies,[252-256] but the effect may be limited to the obese or diabetics. Particles also increase homocysteine in the elderly.[228, 257]

Again, intervention trials in humans have confirmed that these epidemiological results are indeed causal. People randomly assigned to receiving an air filtering device for their home with a true filter or a sham filter were assessed for blood markers of inflammation and oxidative stress. The people getting the sham filter had higher concentrations of CRP (a marker of inflammation) in their blood than those getting the true

filter. They also had higher levels of 8-OHdG, a marker of oxidative damage to DNA[258].

Inflammation can also inhibit immune defenses against infections. Pneumonia commonly results from a bacterial infection after defenses are weakened by a viral one. Sigaud and coworkers examined the effect of particles on this process in animals, and reported that exposure to particles from the ambient air reduced resistance to bacterial pneumonia.[259] Consistent with this, concentrated ambient particle exposure was shown to reduce the ability of macrophages in the lung to kill streptococcal bacteria.[260] And animals infected with Strep pneumonia and exposed to concentrated particles showed doubled bacterial levels and doubled lung area infected compared to control animals breathing filtered air.[194]

These effects are seen at very low exposures. For example, Busso exposed rats for three months to either unfiltered outdoor air with $PM_{2.5}$ (mean=10.6 µg/m^3) concentrations well below EPA's Ambient Air Quality Standard, or outdoor air with the particles removed. Their lungs were examined and the exposed rats showed more evidence of lung inflammation (increased white cells moving into lung tissues, reduced interior size of alveoli, etc.) and DNA damage compared to the control rats[261].

Coagulation/Thrombosis pathway

Both animal and controlled human exposure studies have demonstrated that ambient particles can increase pro-thrombotic (i.e., clot forming) activity and even induce thrombosis in acute exposures. Thrombosis produces a range of adverse outcomes including embolisms, heart attacks, and, on a chronic basis, increased atherosclerosis.[262-264] There is recent epidemiology that supports this finding. Baccarelli and coworkers reported an association of airborne particles with decreased clotting time,[265] as well as the risk of deep vein thrombosis.[266] A further study reported traffic pollution was independently associated with deep vein thrombosis[267]. Air pollution was also associated with changes in global coagulation parameters in 1,218 individuals from the Lombardia Region in Italy (60). And most recently, we examined all admissions of Medicare participants in the Northeastern United States for Deep Vein Thrombosis, using our satellite remote sensing data to estimate $PM_{2.5}$ exposure at every ZIP code in the U.S., and reported strong associations of both short-term and long-term exposure and admission rates, with long term exposures showing a larger effect[268].

These studies are consistent with the results of a controlled exposure study of humans to diesel particles, which reported increased ST depression (the depression of the ST segment of an electrocardiogram, which is a sign of ischemic heart disease) and alterations in fibrinolytic capacity (the ability to break up clots that have formed in blood

vessels).[263] Further support comes from a recent study of almost 58,000 women in the Women's Health Initiative. They found that $PM_{2.5}$ was associated with a 4 percent increase in the risk of an ST abnormality on the electrocardiogram and a 5 percent increase in the risk of a T wave abnormality.[269] ST abnormalities are markers of ischemia.

Suwa et al. have demonstrated that exposure to particles increases plaque formation and decreases plaque stability.[206] Other studies have also shown thrombosis in animals exposed to particles.[264, 270] Such changes increase the risk of a heart attack.

Ischemia

A number of studies have directly linked particle exposure with ischemia (reduced blood supply to the heart). Wellenius exposed dogs to either filtered air or concentrated air particles, followed by a temporary blockage of the coronary artery. The animals exposed to particles experienced greater ischemia than those exposed to filtered air.[271, 272] Similarly, human volunteers exposed to particles manifested myocardial ischemia, and impaired ability to dissolve clots.[263] And a follow-up study of a registry of patients who underwent coronary artery catherization in Utah found an association between particles and ischemic events.[199]

A standard measure of ischemia derived from electrocardiogram patterns is ST segment depression. Recent studies of subjects undergoing repeated examinations have found that particles are associated with

increases in ST segment depression in vulnerable populations.[273-275] This was also seen in human volunteers experimentally exposed to particles. In addition, Kuenzli et al. have reported an association of particle exposure with chronic atherosclerosis, the major risk factor for ischemia.[276] A follow-up study confirmed the previous report that particle exposure was associated with narrowing of the carotid artery.[277] Other studies have also reported associations of particles with various markers of chronic atherosclerosis.[278-281]

Blood Pressure Changes

Elevated blood pressure (BP) predicts cardiovascular morbidity and mortality, including heart attacks, and reductions in BP have been shown to reduce risk. Many studies have observed positive associations between ambient air pollutants and BP,[282-291] although other studies have failed to find associations or have even detected negative associations between ambient air pollutants and BP.[292]

There is evidence of both immediate effects[293] and delayed effects over several days.[289] Providing a mechanistic link relating particles to blood pressure, endothelin-1, which modulates systemic vascular tone and blood pressure, is known to increase in response to reactive oxygen species,[294] which are elevated by particles (as noted above), and endothelin-1 has also been shown to increase directly in response to urban PM.[288, 295-300] Li and colleagues found that losartan, a blood pressure

lowering medication that is an antagonist of angiotensin II type 1 receptors, inhibits the vasoconstriction effect of urban particles on human pulmonary artery endothelial cells.[301] In addition to its effects on blood pressure, angiotensin II is also a proinflammatory mediator,[302] raising the possibility that the local renin-angiotensin system and systemic inflammation may be interrelated components of the cardiovascular system's response to ambient particles and the oxidative stress they induce.

Controlled human exposure studies have recently indicated that airborne particles are associated with acute changes in blood pressure. One study reported an association between arterial diameter and particle exposure.[303] A follow-up study reported a direct association with blood pressure.[293] This is supported by observational epidemiology studies in panels of subjects in Germany and the United States.[289, 292] Another study looked at the change in blood pressure from sitting to standing and found that $PM_{2.5}$ was associated with these changes and that the effects were modified by genes related to pathways of blood pressure control.[285] Further, there is evidence that prenatal exposure to particles increases newborn children's blood pressure.[304] While chamber studies randomly expose subjects to particles or filtered air for a few hours, a number of studies have been able to use randomized controlled trials that reduce exposure in the treated population to look at longer term exposures. McCracken and coworkers randomized houses in rural Guatemala that initially used

unvented open fires for cooking to either receive a chimney stove (intervention group) or not (control group). Women over age 45 who cooked were examined in both groups, and a significantly lower blood pressure was found in the intervention group.[305] At the end of the trial, the control group was given the same stove, and the investigators returned to measure blood pressure in the controls, using a pre-post design. Again, stove intervention was associated with a reduction in blood pressure.

Two hundred homemakers in Taiwan were randomly assigned to receive a functional particle filter in their house, or an identical filtering device but with the actual filter element removed. Six home visits were conducted per year, and blood pressure was measured. The participants with the real filters had lower blood pressure than those with the sham filters, causally demonstrating that particle exposure increases blood pressure[258].

A shorter-term intervention trial was undertaken in Beijing. Subjects walked the streets for two hours twice, once wearing a particle-filtering mask. Blood pressure was measured continuously during the two 2-hour walks. Blood pressure was lower when wearing the filter.[306]

More recently, epidemiology studies have examined the association of long-term exposure to particles and blood pressure. I showed that long-term exposure to particles was associated with higher blood pressure in the elderly[307]. Subsequently, a large study of a cohort of over 43,000 women reported an association between $PM_{2.5}$ exposure and increased blood

pressure[308]. Another cohort study followed 35,000 participants in Ontario from 1996 until 2010 and reported that $PM_{2.5}$ increased the risk of developing high blood pressure[309]. Frighteningly, a study examining freshman college students in California demonstrated that **prenatal** exposure to $PM_{2.5}$ was associated with increased stiffness of the carotid artery.[310]

Electrical Control of the Heart

One potential pathway for the cardiovascular effects of particles is to change the autonomic nervous system, which affects both blood pressure and the electrical control of the heart. The lung contains nerve endings from the autonomic nervous system, and stimulation or irritation of these nerve endings has been shown to have cardiovascular effects. One measure of such effects is from studies that have looked at repeated visits of subjects, where electrocardiograms were measured at each visit. Electrocardiogram changes in these studies have been related to particles in the air.[226, 252, 311-322] Moreover, animal studies, and controlled human exposure studies, have confirmed such results.[319, 323-326] These studies are important because the types of electrocardiogram changes associated with particle exposure have been shown to increase the risk of death. Note that this applies to both short-term and long-term changes in these patterns.

Several other changes in electrocardiograms that are associated with risk of death have also been shown to be produced by particulate air

pollution. These include T-wave Alternans,[327] ST segment depression,[273-275] and QT prolongation.[253, 326, 328, 329] Importantly, the same randomized trial of a stove intervention in Guatemala that produced reductions in blood pressure also produced reductions in ST segment depression.[330]

Endothelial Pathway and Atherosclerosis

The endothelium is the lining of the arteries, and it is not merely the coating of a tube carrying fluid. It is biologically active. When stressed, endothelial cells can activate clotting mechanisms, recruit inflammatory cells, which can disturb atherosclerotic plaques that have previously formed, and impair clot-dissolving functions. Acutely, these can result in ruptures of the plaques, which trigger heart attacks; more chronically, they can result in atherosclerosis. Particles in the air affect these processes. For example, Suwa and coworkers showed in animal models that particles increase the rate of growth of plaques, and decrease stability, increasing the risk of a rupture.[206] In general, indicators of endothelial dysfunction have been shown in epidemiologic,[251, 263, 297, 331-336] toxicological,[223, 295, 301, 337-341] and controlled human exposures.[218] One such exposure was an intervention trial of air filtration for elderly adults, which reduced particles levels and improved endothelial function.[342] These finding are quite consistent with the findings that particles are associated with heart attacks and sudden deaths.

A study of mice genetically prone to atherosclerosis and on a high-fat western diet exposed to concentrated particles from the outside air showed that the particle exposure leads to more atherosclerotic plaque and increased macrophages and tissue factor in the plaques, which reduce plaque stability and increase the risk of a heart attack.[343] An earlier study, done in the northern suburbs of New York with low particle concentrations and little traffic pollution, exposed animals to concentrated particles and again reported increased plaque and increased vasoconstriction in mice genetically prone to atherosclerosis.[344] A more recent study, using a different mouse model of atherosclerosis documented that particle exposure increased oxidation of low-density lipoprotein (making it much more dangerous), increased the thickness of the arterial wall, and promoted plaque growth and instability.[345] Yet another study looked at gene expression in arterial tissue of animals following exposure to filtered air or particles. They reported clear signs of hundreds of genes changing their expression pattern after particle exposure, including increases in the inflammatory and cell proliferation (needed to thicken arterial walls and grow plaque) pathways.[346]

Again, the human studies of atherosclerosis by Kuenzli cited above, which used ultrasound measurements to show particles produced narrowing arteries, have been confirmed by other studies. For example, a study in Germany has confirmed this association of $PM_{2.5}$ with carotid artery thickening.[280] A novel study used the MESA cohort and took advantage of the

fact that the blood vessels in the eye are readily visible in the retina. Special eye examinations used retinal photography and calculated how narrow the retinal vessels were. Investigators used a spatial model to estimate $PM_{2.5}$ at the homes of every subject, and reported that going from the 25^{th} percentile to the 75^{th} percentile of $PM_{2.5}$ exposure was associated with as much a narrowing of the blood vessels as seven years of aging.[279]

I conclude that experimental exposure of animals to particles results in both more hardening of the arteries and decreased stability of the plaques; that multiple studies of human populations have demonstrated more hardening of the arteries in people more exposed to particles; that this is confirmed by experimental studies (air filters) in humans, and this combination is clear evidence that the epidemiologic evidence showing particles increase the risk of heart attacks is causal.

Other Mechanisms: Epigenetics, Biological Aging, and Metabolomics

DNA Methylation

There are a number of other important physiologic pathways that are disturbed by particulate air pollution. Since the processes cited above, such as oxidative stress, inflammation, decline in lung function, hardening of the arteries, etc. are processes that occur even in the absence of air pollution exposure, and are associated with aging, it makes sense to consider whether particles are associated with biological aging itself. In addition, most bodily processes are controlled by the proteins

produced by our genes, and epigenetics is the study of changes to chromosomes that do not change the genetic code, but rather control the expression of genes (i.e., when and how much they are turned on or off). Changes to these processes also happen with aging, and are thought fundamental to the aging process and the development of disease. Particulate air pollution has been shown in multiple studies to interfere with that process.[347-349] Some of these same epigenetic changes have been associated with heart and lung disease.[350-354]

For example, we showed that particles were associated with changes in DNA methylation (one of the key control mechanisms for gene expression) in genes related to the MAP kinase inflammatory response[355]. Another study developed a novel multivariate statistical method for analyzing DNA methylation scores and showed that exposure to particles was associated with DNA methylation at several genes related to Asthma[356]. Recently, we conducted a genome wide scan of DNA methylation changes associated with $PM_{2.5}$ in two cohorts, and reported several genes that were affected[357].

DNA methylation age is novel approach to define biological aging separately from chronological age. Horvath developed a score by looking at how DNA methylation at ~500,000 locations across the genome predicted chronological age in a selected group of healthy people. Using machine learning techniques, he identified a set of 353 locations that were responsible for most of the predictive power. Subsequent studies in more general populations have established that, controlling for chronological

age, this methylation age variable predicted cancer incidence and mortality[358]. Another study showed that methylation age predicted death from all causes[359]. Hence, this has been established as a marker of biological aging distinct from chronological age, and is interpreted as reflecting the extent to which one has aged less or more than the calendar would suggest. Therefore, we examined whether particles are associated with accelerated aging in the population, by looking at their association with this measure of biological age. We showed that annual $PM_{2.5}$ concentration was associated with increased DNA methylation age in the Normative Aging Study[360]. Each 1 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations was associated with an increase of 0.51 years in biological age.

Mitochondrial Function

Mitochondria are organelles in the cells of eukaryotes (plants and animals) that provide the basic energy metabolism for that cell. They take in glucose, and produce other compounds such as adenosine triphosphate, that provide energy for the cellular processes. They appear to be derived from archaic bacteria, which entered into a symbiotic relationship with eukaryotes, and have their own DNA. What they do not have, however, is their own DNA repair mechanism. In contrast, our cells have multiple repair mechanisms to excise and replace damaged pieces of DNA. Consequently, DNA damage is a serious issue for mitochondria. This is worsened because the energy metabolism that they provide involves the

generation of reactive oxygen species, which, if not controlled, can damage the DNA of the mitochondria. Consequently, as we age, the mitochondria in our cells accumulate more and more damage, which is considered an important feature of biological aging. One response of a cell to impaired mitochondria is to make more copies of them (cells have tens to hundreds each) in an attempt to compensate for the damaged ones, and mitochondrial DNA copy number is an established indicator of the extent of mitochondrial damage. Under certain stressors, DNA copy number can fall, however. For example, Diabetes is associated with lower DNA copy number. In light of this, we have examined whether mitochondrial copy number is associated with particle exposure. In Hou, we showed that occurred, with higher particle exposure associated with increased copy number as cells struggled to cope with increased mitochondrial damage[361]. However, in a randomized exposure to $PM_{2.5}$ or filtered air, particle exposure depleted 11% of Mitochondrial DNA and induced methylation changes in genes responsible for mitochondrial oxidative energy production[362].

Telomere Length

Another key indicator of biological aging is telomere length. Telomeres are caps at the ends of each chromosome, which protect them when the DNA is unraveled for cell division. Each cell division, however, tends to reduce the size of the caps. When they are completely lost, the DNA tends to be damaged, and the cells can no longer successfully reproduce.

Since the ability to reproduce cells to replace damaged ones is essential for life, this is thought to be a principal factor limiting life expectancy. Hence discovering a substance that reduces telomere length indicates it is a likely risk factor for reduced life expectancy. Telomere length is most easily measured in white blood cells, which introduces a complication. Wounds, infections, or systemic inflammation can increase demand for white blood cells, causing the bone marrow to produce more of them. This will lower the average age of white cells in the blood, which can paradoxically result in longer average telomere length. In a study of workers in Beijing, we found we found that telomere length in fact increased with higher exposure to particles on the same day, but was shortened in response to higher exposure to particles over the preceding two weeks[363]. This loss of telomere length with longer exposure is precisely what we have found for particulate air pollution in both occupational cohorts[364] and in an elderly population sample[365]. A study in an elderly population of never smokers in Europe confirms this, with $PM_{2.5}$ demonstrated to reduce telomere length[366].

MicroRNA

Another mechanism by which the expression of genes is controlled involves microRNA. MicroRNAs are very small (~22 base pairs) strips of RNA. Unlike the much longer messenger RNA, they do not contain the code for making proteins. Messenger RNA duplicates the code for a protein from

the DNA in a gene and carries it to a Ribosome, where the protein is assembled according to the code. MicroRNAs can bind to the messenger RNA and prevent that last stage of the process. Hence, they are used in the body to limit production of proteins. Again, inappropriate amounts of microRNAs can lead to impaired physiology and disease. While microRNAs produced in a cell generally act in that cell, there is a process whereby they are packaged in small pieces of cell membrane material, called vesicles, and released from the cell to travel through the bloodstream to effect protein production elsewhere. These extra-cellular vesicles (EVs) containing microRNA are now recognized as a key part of cell to cell communication, which in turn influences key processes such as inflammation. A study of 1,630 people found that particle exposure resulted in increased release of extracellular vesicles, particularly by monocytes and macrophages. In turn, the particle exposure was associated with reductions in the amount of nine microRNAs in these vesicles which are related to cardiovascular disease, and five of them were mediators of the particle induced increase in fibrinogen, a major clotting factor[367].

Metabolomics

Thousands of chemicals are produced in the human body in order to perform its functions, and it is increasingly clear that disturbances to the patterns of these multiple metabolites of normal bodily functions are indicators of disease, precursors to disease, and potentially causes of

disease. Metabolomics seeks to identify these patterns of change in response to exposure and to disease. A recent study by the Dutch National Institute for Public Health and the Environment exposed people to ambient air pollution in different locations in the Netherlands, and took blood samples 2 hours before exposure, 2 hours after exposure, and 18 hours after exposure. Diet and activity of the subjects were standardized by the researchers before the exposure took place. Several standard health outcomes (lung function, fibrinogen in blood (a clotting factor), etc.) were measured to see if the metabolites mediated the effect of air pollution on health measures. Their analyses controlled for the levels of the metabolites before exposure, to capture the baseline status of each participant, and corrected for the statistical issue that arises when one examines thousands of compounds, to wit, the increased risk of finding something by chance. Notably, they found that sulfate particles were associated with lower lung function, more clotting factor, and with dozens of metabolic changes 18 hours after exposure[368]. Soot particles were associated with lower lung function, more inflammation, and several metabolites two hours after exposure.

III.A.7. CONCLUSIONS ABOUT PARTICLES

In summary, particles in the air are associated with increased rates of deaths, heart attacks, hospitalizations for pneumonia, blood pressure, impaired cognition, and other serious health impairments. There is no

evidence for a threshold for these effects, which means that any incremental exposure is associated with incremental deaths, heart attacks, etc. in the general population. Hence, any emissions of particle precursors cause serious harm to human health, including early deaths, and any reduction in emissions will have health benefits. For these reasons, it is my opinion that people living in locations whose particle concentrations are increased by excessive NOx emissions from the Navistar engines will suffer adverse health effects.

The benefits of reversing these excess emissions will occur quickly. For example, as described in above, I reanalyzed the Harvard Six City Study data, and asked whether the increased mortality seen with higher $PM_{2.5}$ depended on lifetime exposure, or only more recent exposure. The results indicated that it was exposure in the previous two years that was responsible for the increased mortality risk[93]. The analysis of the Nurses Health Study by Puett and coworkers reported similar findings—the effects of $PM_{2.5}$ on mortality are predominantly from the previous two years[23].

We see this for other outcomes as well. For example, Currie and Walker took advantage of the introduction of Easy Pass (E-Zpass) on toll roads in New Jersey to study the importance of traffic pollution exposure on adults[369]. Traditional toll plazas, which generated more pollution because of the congestion they caused, were replaced with Easy Pass toll plazas, but not all at the same time. The construction company determined the timing, based on convenience, and independent of any predictors of

health. Hence, it represents effectively a randomized trial of lowering traffic pollution on health. Among mothers giving birth during the transition and living near toll plazas, the introduction of EZPass reduced prematurity by 10.8 percent and low birthweight by 11.8 percent compared to births in the neighborhoods near plazas that had not yet received E-ZPass. Similarly, they followed mothers who lived near toll plazas over time and compared siblings born before and after the adoption of E-ZPass. They found that prematurity was 1.4 percent lower and low birthweight was 1.1 percent lower in siblings born after the change compared to those born before. Thus, health effects changed within a year of the change in exposure.

Another study looked at deaths heart disease before, during, and after the Beijing Olympics. China shut many factories in the area surrounding Beijing during the Olympic Games to lower air pollution concentrations. Heart disease deaths also dropped during the period, and rebounded once the pollution resumed[370]. Again, the two Pope studies of natural experiments, the copper smelter and steel strike studies, showed that abrupt changes in pollution produced abrupt changes in death rates, and in hospital admissions of children for asthma and pneumonia.

The southern California Children's cohort study reported that $PM_{2.5}$ was associated with lower lung function growth in children. But, as described above, if they moved to a lower pollution location, their rate of growth increased again[121]. Again, Giroux and coworkers (2001) looked at

asthmatic children spending the summer in the city vs. those in a national park and reported spending the time in the national park dropped the concentrations of exhaled NO, a marker of inflammation in asthmatic lungs, to half its value of those remaining in the city[132].

Hence if we reduce exposure, the evidence indicates that most of the health benefits will accrue within one to two years, and that requiring such reductions to make up for prior violations will produce immediate benefits to the downwind populations. In addition, of course, these studies of abrupt changes in exposure and health demonstrate the causality of the associations.

III.B. Health Effects of Ozone

Background on Ozone

Ozone is a ubiquitous pollutant but is not emitted directly. Rather, it is formed in the atmosphere by the chemical reaction of other substances. For example, hydrocarbons in the air, from manmade or natural sources, react with nitrogen oxides, the pollutant emitted in excess of standards by Navistar, in the presence of light and heat to form ozone. The basic reactions are:

$NO_2 + VOC + sunlight \rightarrow NO + O + VOC$

$O + O_2 \rightarrow O_3$

Carbon monoxide, another combustion byproduct, also plays a role in ozone formation. Since the chemical reaction takes time to complete, ozone concentrations are not limited to, or even highest in, the locale where the precursors were emitted, but can travel thousands of kilometers, exposing new populations as it travels. Ozone itself is a powerful oxidant, capable of harming living organisms.

Ozone and Health

III.B.1 The Scientific Consensus

In its recent ISA for ozone, EPA states:

"The last review concluded that there was clear, consistent evidence of a causal relationship between short-term exposure to O3 and respiratory health effects. This causal association was substantiated in this ISA by the coherence of effects observed across controlled human exposure, epidemiologic, and toxicological studies indicating associations of short-term O3 exposures with a range of respiratory health endpoints from respiratory tract inflammation to respiratory emergency department (ED) visits and hospital admissions (HA). Across disciplines, short-term O3 exposures induced or were associated with statistically significant declines in lung function. An equally strong body of evidence from controlled human exposure and toxicological studies demonstrated O3- induced inflammatory responses, increased epithelial permeability, and airway hyper-responsiveness. Toxicological studies provided additional evidence for O3-induced impairment of host defenses. Combined, these findings from experimental studies provided support for epidemiologic evidence, in which short-term O3 exposure was consistently associated with increases in respiratory symptoms and asthma medication use in asthmatic children, respiratory-related hospital admissions, and asthma-related ED visits. Although O3 was consistently associated with non-accidental and cardiopulmonary mortality, the contribution of respiratory causes to these findings was uncertain. The combined evidence across disciplines **supports a causal relationship between**

short-term O3 exposure and respiratory effects." (emphasis in original).

For cardiovascular effects, the ISA concludes that there is "likely to be a causal relationship" (p 1-5). The ISA also states

"Recent multicity studies and a multi-continent study have reported associations between short-term O_3 exposure and mortality, expanding upon evidence available in the last review (see Section 6.6). These recent studies reported consistent positive associations between short-term O_3 exposure and total (non-accidental) mortality, with associations being stronger during the warm season, when O_3 concentrations were higher. They also observed associations between O_3 exposure and cardiovascular and respiratory mortality. These recent studies also examined previously identified areas of uncertainty in the O_3-mortality relationship, and provided additional evidence supporting an association between short-term O_3 exposure and mortality. As a result, the current body of evidence indicates that there **is likely to be a causal relationship between short-term exposures to O_3 and total mortality.**"

(emphasis in original). It similarly finds that long-term exposure to ozone is likely to have respiratory effects.

The World Health Organization, in setting its guidelines for ozone, stated:

"These latest time-series studies have shown health effects at ozone concentrations below the previous guideline of 120 μg/m^3 but without clear evidence of a threshold. This finding, together with evidence

from both chamber and field studies that indicates that there is considerable individual variation in response to ozone, provides a good case for reducing the WHO AQG for ozone from the existing level of 120 μg/m^3 to 100 μg/m^3 (daily maximum 8-hour mean). "

In a letter to Administrator McCarthy, commenting on EPA's recent proposed ozone standard, a wide range of medical associations expressed their opinion that ozone was harmful at current exposure levels, and among other things, caused early death. The full text of their letter is in Appendix A, selections are below:

"American Academy of Pediatrics • American College of Preventative Medicine • American Heart Association • American Lung Association American Medical Association • American Public Health Association • American Thoracic Society • Asthma and Allergy Foundation of America • Children's Environmental Health Network • National Association of County and City Health Officials • National Association for Medical Direction of Respiratory Care • Health Care Without Harm • Trust for America's Health

Re: EPA Docket I.D. No: EPA-HQ-OAR-2008-0699

Dear Administrator McCarthy:

As national organizations representing medical societies, public health and patient advocacy organizations, we write to provide comments to the U. S. Environmental Protection Agency on the proposed Ozone National Ambient Air Quality Standards. Our organizations

appreciate and would like to express our support to the EPA for moving forward to update the current ozone standard, and welcome this opportunity to provide input to this process, which we hope will result in a standard that is better protective of public health. Our organizations urge you to select a level for the primary health standard that will meet the Clean Air Act requirement to protect the health of the public with an adequate margin of safety: 60 parts per billion (ppb)."

And

"The list of populations who risk demonstrated harm from ozone pollution has grown significantly from the previous review. Children, people with asthma and other lung diseases, seniors, outdoor workers and people who have low socioeconomic status have long been shown to be vulnerable to ozone. Newer evidence shows some otherwise healthy adults are especially sensitive to ozone exposure because of limitations in some nutrients and certain genetic variants. In addition to these groups, the EPA's Integrated Science Assessment has documented evidence that suggests increased risk to fetal development and to cardiovascular harm (EPA, Integrated Science Assessment, 2013). Health-based standards must be set at levels that will protect all people, but particularly these sensitive groups. Ozone poses a grave threat to public health at levels well below the current standard. The current standard of 75 ppb fails to meet the

requirements of the Clean Air Act. Clinical and epidemiological studies have repeatedly shown that breathing ozone can threaten life and health at concentrations far lower than the 75 ppb 8-hour average standard. Extensive, public reviews of the large body of evidence by EPA's independent science advisors, the Clean Air Scientific Advisory Committee (CASAC), and by EPA staff scientists have confirmed that the 2008 primary ozone standard is set at a level that is too weak to protect public health. In fact, three successive CASAC panels -- each under different leadership -- have reached the same conclusion: the 2008 standard should not be retained."

And

"The scientific and medical understanding of the mechanisms by which exposure to ambient ozone pollution harms human health has grown considerably stronger since 2007. The EPA evaluated 1,000 new studies in the current review, studies that have been published since the completion of the 2006 Criteria Document. These studies inform our understanding of the health impacts of ozone at low concentrations. Multiple chamber studies provide robust evidence of harm to healthy adults down to 60 ppb. Adding to previous research by Adams (2002) and Adams (2006), both Brown et al (2008) and Kim et al (2011) provide still more evidence that exposures down to 60 ppb can reduce lung function and cause inflammation that meets the American Thoracic Society's criteria for judging adversity. The subjects in these

chamber studies were healthy young adults -- not children, the elderly, or people with asthma who are more susceptible to ozone. The chamber studies establish solid evidence that concentrations above 60 ppb would provide significant risk not only to many healthy adults, but most critically, to susceptible populations, including children, seniors and people with asthma and other chronic lung diseases."

And

"In addition to the strong evidence of increased morbidity from ozone down to 60 ppb, multiple well-reviewed studies had identified a new, strong association with premature death, with no discernable threshold, that made the risks to the large, vulnerable groups even graver."

And

"Research not only confirms the previous conclusions about ozone's impact on human health, but adds to and clarifies the impact on multiple physiologic systems, including respiratory and cardiovascular. Examination of long-term exposure has identified outcomes beyond the traditional concerns to include the central nervous system and reproductive and developmental effects. The growing evidence of effects associated with breathing ozone for longer periods adds to the urgency to set the most protective standard now to reduce those exposures. "

And

"Since the previous review large studies examining exposures in multiple cities and continents have shown the consistent and pervasive threats to respiratory health. New studies confirm the impact on children with asthma. Multiple studies demonstrated increased pulmonary inflammation (Berhane et al., 2011; Khatri et al., 2009; Barraza-Villerreal et al, 2008), and increased risk of hospital admissions (Silverman and Ito, 2010; Strickland et al., 2010). Several large studies looking at single cities and multiple cities confirm that breathing ozone increases the risk of hospital admission and emergency department visits for respiratory conditions (Katsouyanni et al, 2009; Lin et al., 2008a; Wong et al., 2009; Darrow et al., 2011); Stieb et al., 2009). Multiple- and single-city studies showed increased risk of respiratory hospital admissions and emergency department visits in cities that met the current ozone standard of 75 ppb (Cakmak et al., 2006; Dales et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009) or where most cities would have met standards set at either 65ppb or 70 ppb (Cakmak et al., 2006; Katsouyanni et al., 2009; Stieb et al. 2009). The American Thoracic Society summarized some of the new studies in the attached editorial in the American Journal of Respiratory and Critical Care Medicine advocating EPA adoption of a standard of 60 ppb (Rice, et al., 2015). 'Highlights of this new body of evidence include a study of emergency department visits among children aged 0 to 4 in Atlanta,

which found that each 30 ppb increase in the 3-day average of ozone was associated with an 8% higher risk of pneumonia and a 4% higher risk for upper respiratory infection(5)[Darrow et al 2014].'"

And

"Perhaps of greatest concern, there is now stronger evidence of increased mortality in association with ozone (17–19)[Peng et al 2013, Romieu et al 2012,Zanobetti and Schwartz 2008], particularly among the elderly and those with chronic disease(20, 21)[Medina-Ramon and Schwartz 2008, Zanobetti and Schwartz 2011]."

And

"Evidence is accumulating about the cardiovascular effects of ozone, with the strongest evidence for increased risk of premature death. Previous studies have shown adverse associations between ozone exposure and various cardiovascular health endpoints, including alterations in heart rate variability in older adults (Park et al., 2005), cardiac arrhythmias (Rich et al., 2006), strokes, (Henrotin et al., 2007) heart attacks (Ruidavets et al., 2005), and hospital admissions or cardiovascular diseases (Koken et al., 2003). Newer large epidemiologic studies from the U.S. (Zanobetti and Schwartz, 2008b), Europe (Samoli et al., 2009) and Asia (Wong et al 2010) have provided evidence of premature death from cardiovascular effects, including two large studies that confirmed the effect after

controlling for particulate matter exposure (Katsouyanni et al 2009; Stafoggia, 2010)."

And

"A growing body of research raises concerns about longer-term exposure to ozone, particularly during pregnancy. Some toxicological studies warn that ozone may affect development of the pulmonary system and central nervous system. Several large studies in California and Australia point to association of prenatal ozone exposure with low birth weight and impaired fetal growth (Salem et al., 2005; Morello-Frosch, et al. 2010; Hansen et al 2007, Hansen et al 2008;Mannes et al 2005). Low birth weight is linked to increased risk of chronic disease as adults (Rogers et al., 2012; Berends et al., 2012). Central Nervous System Effects Increased research since the last review has expanded evidence of the potential effects on the central nervous system. Toxicological studies provide evidence that short- or long-term exposure to ozone may affect cognitive abilities, such as memory (Rivas-Arancibia et al., 1998), and may produce changes similar to those seen in human neurodegenerative disorders (Rivas-Arancibia et al., 2010; Santiago-López et al., 2010; Guevara-Guzman et al., 2009). "

And

"Short-term increases in ozone were found to increase deaths from cardiovascular and respiratory causes in a large 14-year study in 95

U.S. cities. The relationship between mortality and ozone was evident even on days when pollution levels above 60 ppb were excluded from the analysis. (Bell, et al., 2004). A series of meta-analyses and multi-city studies has documented an increase in premature death following ozone exposures below 75 ppb, particularly among the elderly (Bell, et al., 2005; Levy et al., 2005; Ito et al., 2005). Furthermore, research has focused on controlling for weather variables in assessing the effect of ozone on mortality. A case crossover study (Schwartz, 2005) of more than one million deaths in 14 U.S. cities found that "the association between ozone and mortality risk is unlikely to be confounded by temperature. Multiple new studies have confirmed that ozone causes premature deaths (Zanobetti and Schwartz, 2008b; Samoli et al., 2009; Wong et al 2010) and provided evidence that these deaths occur even after controlling for other pollutants, including particulate matter (Stafoggia, 2010; Katsouyanni et al., 2009). Of special concern the risk of premature death from ozone showed up more frequently in communities with higher unemployment or that had a higher percentage of Black/African-American population, as well as in individuals who were Black/African-American or who had lower socioeconomic status. (Median-Ramón and Schwartz, 2008). EPA needs to ensure the strongest, most protective standards are in place to prevent this deadly pollutant from threatening the lives of thousands of Americans."

Sincerely,

American Academy of Pediatrics

American College of Preventive Medicine

American Heart Association

American Lung Association

American Medical Association

American Public Health Association

American Thoracic Society

Asthma and Allergy Foundation of America

Children's Environmental Health Network

Health Care Without Harm

National Association of County and City Health Officials

National Association for Medical Direction of Respiratory Care

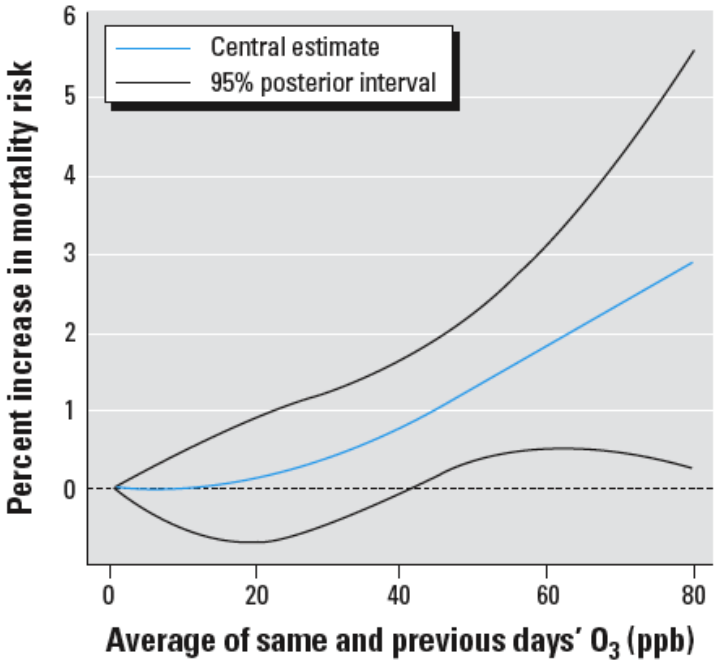
Trust for America's Health."

Hence, the scientific consensus is that ozone kills people, puts them in hospitals for respiratory and possibly other conditions, affects heart disease, induces pregnancy complications, and impairs the health of asthmatics. In addition, as described below, there is a new body of substantial evidence, published since the ISA, that supports those conclusions, strengthens the conclusion about the respiratory effects of long-term ozone exposure, and finds that long-term exposure to ozone is associated with increases in long-term mortality rates, including from

cardiovascular disease. I concur in the consensus, and also believe the new evidence for long term mortality effects.

III.B.2. Ozone kills People

While studies have for decades reported adverse responses to ozone, a highly oxidizing gas, studies demonstrating that ozone exposure might hasten deaths have been more recent. The first large scale report came from Europe, where seven cities were studied using identical methods and the results combined.[371, 372] Since then, a number of studies have reported similar results in both the U.S. and Europe, including three large meta-analyses commissioned by the U.S. EPA.[373-375] Moreover, a large multi-city study has found no evidence of a threshold down to very low levels.[376] The figure below is reproduced from that paper. Again, this means any increase in exposure above 20 ppb will result in increased deaths.



Source: Bell et al. (2006).

Confounding

The major concern of observational epidemiology studies is that some other exposure, correlated with the exposure of interest, may explain the observed association, which is not causal, but due to that correlation. For another exposure to confound studies of short-term changes in ozone and daily deaths, it must co-vary with ozone over the same timescale. The two obvious candidates for such a confounder are temperature and other secondary pollutants.

Polluting sources do not directly emit ozone. It is produced by chemical reactions in the atmosphere, and those reactions are driven by sunlight and temperature. Hence ozone co-varies with temperature. All

studies of the effect of ozone on daily deaths have therefore controlled for temperature. However, the association of temperature with death is highly nonlinear; with heat wave conditions associated with much larger increases in deaths than temperatures just a few degrees cooler. How can we be sure that those studies correctly captured that relation, and that the ozone association is not due to ozone capturing the remaining effect of temperature?

I addressed this in an analysis of over one million deaths in 14 cities.[377] Rather than examine the correlation between daily ozone and daily deaths, I converted the analysis into a case-control study. Using a variant called a case-crossover analysis (discussed above, regarding particle studies), I matched each decedent with themselves, on a control day in the same month of the same year that they died, which also had the same temperature (rounded to degree). This matching controlled for season and time trend, by choosing a control day in the same month and year as the date of death. Since the temperature was the same on the control day as the case day, temperature could not explain which day the death occurred on. I then compared the ozone levels on the two days to see if they predicted which day was the date of death. I found the same association with ozone that I found analyzing the data using the more traditional time series analysis. This indicates that confounding by temperature does not explain the observed ozone-mortality association.

The same process, (chemical reactions driven by light and heat), which produces ozone also produces other secondary pollutants (secondary because they are not what were primarily emitted). Among these are sulfate particles, from the reaction of sulfur oxides with ammonia, and organic particles, which, like ozone, derive from reactions of hydrocarbons. These pollutants are rarely measured, and hence previous studies have not controlled for them.

To address this, Franklin and Schwartz turned to the U.S. EPA's speciation monitoring network.[378] Unfortunately, this network has only been operating since 2000, usually monitors only 1 in 3 or 1 in 6 days, and only measures particles, and not other oxidant gases. Nevertheless, using data from 18 cities with speciated particle measurements, we showed that control for nitrate particles or organic carbon particles did not change the estimated effect of ozone on mortality. In contrast, control for sulfate particles reduced the estimated ozone effect by about 25%, although the confidence intervals included the possibility of no reduction. Hence some of the effect attributed by past studies to ozone may have been due to sulfate particles, but most was not, and organic and nitrate particles do not appear to be confounders.

A more recent studies have confirmed the association of short-term exposure to ozone and daily death rates in the entire Medicare population of the US[379]. This study covered 62 million people across the entire country.

Harvesting

One possible explanation of the observed associations is that they are causal, but that only extremely sensitive individuals, who are on the brink of death, are affected by this exposure. If ozone is merely bringing forward deaths among people who would have died in the next week anyway, the public health impact of the observed ozone-mortality association is much reduced. Recently, we addressed this question in a large, multi-city study.

If ozone's primary effect is on the death rate from the risk pool, and deaths were only being brought forward by, e.g., seven days, then, *ceterus paribus*, we would expect a negative correlation between ozone exposure today and deaths a week from now. Zanobetti and Schwartz used this insight to look at the correlation between ozone levels and death counts in 48 U.S. cities for time periods up to 21 days after exposure.[380]

We found no negative correlation existed between ozone and mortality up to 21 days later, and that the positive association persisted over several days but fell to zero within a few days. The overall effect of ozone over the period was an increase of 0.5% in daily deaths (95%C.I.: 0.05-0.96) per 10 ppb increase in 8-h average ozone, compared to an increase of 0.3% (95%C.I.: 0.2-0.4) when only one day of ozone exposure was considered. Hence, the deaths associated with ozone are not just being brought forward by a few weeks, and previous studies may have

underestimated the overall effect of ozone on mortality by just considering the effect of the ozone on deaths the same day.

III.B.3 Long Term Ozone Exposure and Mortality

Long-term exposure to ozone and mortality is a more serious issue since such associations could not be detected without significant reductions in life expectancy. Until recently, only one cohort study has examined this question, and it reported an association between long-term average ozone exposure and long-term average mortality rates from respiratory disease.[381] More recently, we published a paper that examined cohorts of Medicare beneficiaries in multiple cities. In an important distinction from the Jerrett study, we only looked within a city for the association between ozone and life expectancy, eliminating confounding by factors that vary between cities. What we showed was that year-to-year variations of summer ozone concentrations around the trend line for each city were associated with year-to-year variations in mortality rates around the trend lines in that city.[382] In a recent follow-up study, we reported that annual average ozone concentrations were associated with shortened life expectancy in another set of cohort studies that controlled for temperature variability[383].

This literature has now been extended substantially. Turner et al. followed the same ACS cohort as Jerrett, but with additional years of follow-up, and twice as many deaths[49]. In addition, they used greatly

improved exposure estimates based on combinations of land use regression and chemical transport models. They report a significant association of annual ozone and all-cause mortality with a 10 ppb increase in ozone associated with a 2 % increase in annual all-cause mortality rates, and a 3% increase in cardiovascular mortality rates in models controlling for $PM_{2.5}$ and NO_2. Moreover this comes on the heels of another paper, the CanCHEC study[384], looking at a cohort of 2.5 million Canadians, reported essentially identical size of effect on all-cause mortality, and somewhat larger effects for cardiovascular mortality. The Turner paper also includes extensive sensitivity analyses showing no evidence that the ozone effects are confounded by individual or area-based measures of socio-economic status, or of pollution modeling strategy. A key finding in the Turner and CanCHEC studies is that ozone was associated with cardiovascular mortality rates, and not just respiratory deaths. Because so many more people die from heart disease than respiratory disease, even modest increases in risk of those deaths implies a large increase in attributable deaths.

Other recent cohort studies include the study of Di and coworkers where, using the entire Medicare population, we showed that annual average ozone concentrations were associated with increased mortality, controlling for $PM_{2.5}$[385]. Two other studies have examined county level ozone and county level all cause deaths[386], or deaths from chronic lower respiratory disease[387] and reported significant associations.

Supporting this result, another cohort study reported an association between long term ozone exposure and factor VII coagulant activity[388], a chamber study reported that ozone affected fibrinolytic activity[389], and a toxicology study reported that following ozone exposure, isolated coronary vessels exhibited greater basal tone, enhanced susceptibility to serotonin stimulation, and impaired response to acetylcholine[390].

III.B.4 Ozone increases Hospital Admissions and other morbid conditions

Ozone and Asthma

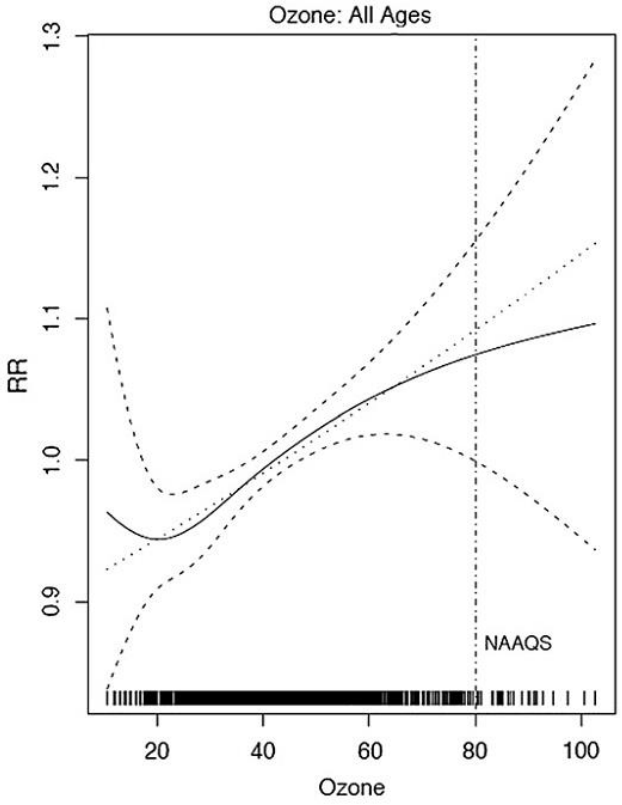
A key finding of the EPA ISA is:

> Collectively, these findings provided biological plausibility for associations in epidemiologic studies of short-term ambient O3 exposure with respiratory symptoms and respiratory-related hospitalizations and emergency department (ED) visits.
>
> . . .
>
> . . . In addition to lung function, ambient O3 exposure has been associated with increases in respiratory symptoms (e.g., cough, wheeze, shortness of breath), especially in large U.S. panel studies of children with asthma (Gent et al., 2003; Mortimer et al., 2000).

(EPA ISA, at 6-1, 6-4).

One of the papers cited by the ISA, by Silverman and Ito, examines asthma hospital admissions in New York City.[391] The figure below, reproduced from the ISA *(Id.*, at 6-123), shows the relative risk of admission vs. ozone

concentration from that study. The relationship continues down to very low levels (20ppb), making it clear that incremental exposure will result in incremental admissions.



Also, ample evidence shows that short-term ozone exposure is associated with decrements in lung functions, increased respiratory symptoms, and lung inflammation.[392] For example, Mortimer examined a large cohort of children with asthma and demonstrated that ozone exposure was associated with increased respiratory symptoms such as shortness of breath, wheeze, and cough. Some studies showed also that the effect is higher in asthmatic or already impaired respiratory function individuals.[393-395]

Ozone and Other Hospital Admissions

Respiratory Admissions

A recent paper examined the association of annual average ozone exposure, rather than daily exposure, on hospital admission rates in the Medicare population of the Southeastern U.S. They reported that, after controlling for $PM_{2.5}$, ozone was associated with increased admissions for pneumonia, chronic obstructive pulmonary disease, and lung cancer[396]. The association with pneumonia and COPD remained when the data was restricted to locations that were always in attainment with the recent ozone NAAQS (70ppb). A study of pediatric emergency room visits in Atlanta, Dallas, and St. Louis reported that ozone was associated with higher rates of emergency visits of children with respiratory illness[397].

Cardiovascular Admissions

The Medicare paper cited above also examined the association of long-term ozone exposure and cardiovascular hospital admissions. We found that ozone was also associated with admissions for stroke and for heart attacks.

Biological Mechanisms

A review of toxicological studies found decreased heart rate, metabolism, blood pressure, and cardiac output when rats are exposed to typical concentrations of ozone. The authors concluded that while only limited experimental evidence addresses the underlying mechanisms of these responses, there is some indication that they may be related to

stimulation of pulmonary irritant receptors and that they may be at least partially mediated via the parasympathetic nervous system.[398]

Other studies showed that the respiratory inflammation may inhibit recovery from infection or produce systemic responses. A recent panel study found that ozone was associated with increased levels of C reactive protein, fibrinogen, 8-hydroxy-2'-deoxyguanosine, plasminogen activator inhibitor 1, and decreased heart rate variability.[399] Two previous papers had also reported decreases in heart rate variability.[400, 401]

The above biomarkers are risk factors for heart disease. And there is more support for this. Other studies reported ozone exposure increase a marker of oxidative stress in the blood (8-isoprostane), of inflammation in the blood (IL8), damaged mitochondrial DNA in the aorta, etc. Many more details are provided in the ISA. Interestingly, these indications of increased inflammation and oxidative stress also are seen in the brain. This is also seen in experimental studies. Devlin exposed twenty-three young healthy people in a randomized crossover fashion to clean air and to 0.3-ppm ozone for 2 hours while intermittently exercising. Blood was obtained immediately before exposure, immediately afterward, and the next morning. Continuous electrocardiogram monitoring began immediately before exposure and continued for 24 hours. Ozone produced a 98.9% increase in interleukin-8 (an inflammatory protein), and a 51.3% decrease in the high-frequency component of heart rate variability, and a 1.2% increase in QT duration (both changes increasing risk of arrhythmia)[402].

Ozone exposure for four hours per day was shown to increase oxidation of the lipids (fats) in the brain, impair learning, and damage the hippocampal region of the brain, where learning occurs[403, 404].

Another recent paper examined the effect of ozone on electrocardiogram patterns in patients in North Carolina[405]. They reported that, controlling for $PM_{2.5}$, ozone exposure was associated with prolonged QT interval, a known risk factor for arrhythmia, increased heart rate, and increased PR interval, which is a predictor of atrial fibrillation and all cause mortality. These associations continued when restricted to exposures below the ambient air quality standards.

A chamber study of ozone exposure in humans demonstrated that ozone exposure increased levels of stress hormones and increased levels of lipids in the blood likely linked to inflammation[406].

These findings of increased markers of systemic inflammation, thrombosis, oxidative stress, and impaired autonomic function support a plausible association with cardiovascular mortality.

III.C. Health Effects of NOx Exposure

There is considerable evidence about the health effects of NO2 as well. I discuss this below. Please note that NO2 is reported in two different units in studies, as parts per billion of air (i.e. out of a billion molecules of air, how many are NO2) and as $\mu g/m^3$, that is, how many

micrograms of NO2 there are in 1 cubic meter of air. 1 ppb of NO2 corresponds to 1.88 µg/m^3.

III.C.1. The Scientific Concensus

EPA has also prepared an Integrated Science Assessment as part of its 2010 setting of the standard for NO2, and updated it in 2016. I here summarize key parts of those conclusions, and then discuss what has changed since their publication.

The 2008 ISA states:

> "The ISA concludes that, taken together, recent studies provide scientific evidence that is sufficient to infer a likely causal relationship between short-term NO2 exposure and adverse effects on the respiratory system (ISA, section 5.3.2.1). This finding is supported by the large body of recent epidemiologic evidence as well as findings from human and animal experimental studies. These epidemiologic and experimental studies encompass a number of endpoints including [Emergency Department (ED)] visits and hospitalizations, respiratory symptoms, airway hyper-responsiveness, airway inflammation, and lung function. Effect estimates from epidemiologic studies conducted in the United States and Canada generally indicate a 2-20% increase in risks for ED visits and hospital admissions and higher risks for respiratory symptoms (ISA, section 5.4)."

EPA has since updated its ISA for NO2. The 2016 version now states:

> "*A causal relationship is determined for short-term NO2 exposure and respiratory effects.*" Executive Summary, p LXXXII.

And:

> "There is likely to be a causal relationship between long-term NO2 exposure and respiratory effects (Section 6.2.9) based on the evidence for development of asthma."

For mortality, the ISA concluded that the evidence for both long-term and short-term exposure was suggestive of a causal relationship.

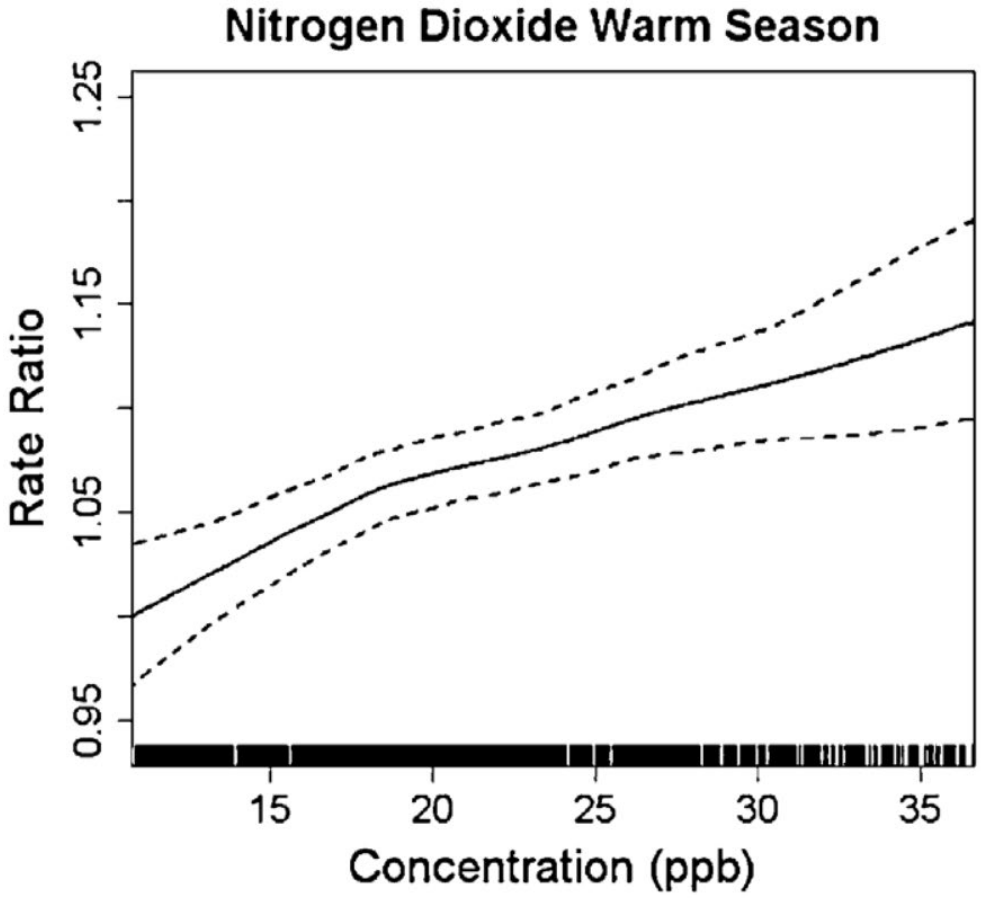
The World Health Organization most recent statement on air pollution (https://www.who.int/airpollution/ambient/pollutants/en/) states:

> "Nitrogen dioxide, mainly emitted by power generation, industrial and traffic sources, is an important constituent of particulate matter and ozone. There is growing evidence that independently, it can increase symptoms of bronchitis and asthma, as well as lead to respiratory infections and reduced lung function and growth. Evidence also suggests that NO_2 may be responsible for a large disease burden, with exposure linked to premature mortality and morbidity from cardiovascular and respiratory diseases."

EPA has cited substantial evidence in the published scientific literature for this conclusion. Importantly they have also addressed the issue of threshold and whether the NO_2-Asthma relationship continues to low concentrations, stating

"Finally, a few recent studies examined whether the shape of the NO_2-asthma ED visit relationship is linear or provides evidence of a threshold. These studies provide evidence of a linear, no-threshold relationship between short-term NO2 exposures and asthma ED visits (Li et al., 2011b; Strickland et al., 2010)." (p 5-92).

For example, the attached figure from the ISA (Fig 5.5, p 5-87) shows the concentration-response curve between the rate of asthma emergency room visits vs. NO_2 concentration, with the reference rate set at the lowest observed concentrations.



Note: ppb = parts per billion. Solid line = locally weighted scatterplot smoothing concentration-response estimates. Dashed lines = twice-standard error estimates. Results are from generalized additive models. Results are presented for the 5th to 95th percentiles of nitrogen dioxide concentrations.

Source: Reprinted with permission of the American Thoracic Society, (Strickland et al., 2010).

Figure 5-5 Concentration-response function for the association between 3-day average (lag 0–2) nitrogen dioxide concentrations and emergency department visits for pediatric asthma in the Atlanta, GA area.

III.C.2. My Opinion

Since the time the ISA was prepared the literature has grown in important ways. A new meta-analysis searched the literature and identified 15 studies that used NO_2 as an exposure and looked at the incidence of lung cancer[407]. They reported a significant association with a 4% increase in

lung cancer rates for a 10 µg/m^3 increase in NO_2. Another study has been published analyzing a large cohort in The Netherlands. They reported a significant association of NO_2 exposure with lung cancer incidence, with a 30 µg/m^3 increment in NO_2 associated with a 29% increase in the risk of lung cancer[25].

Another systematic review and meta-analysis examined the association of short term NO_2 exposure with mortality and hospital admissions[408]. After an extensive literature review, they identified 204 time series studies that had examined the association of NO2 with either daily deaths or hospital admissions. They reported significant associations with multiple outcomes. For example, there were 101 studies that examined the association of NO2 with daily deaths. They found a significant association with deaths from all causes, with a 10 µg/m^3 increase associated with a 0.71% increase in daily deaths (95% Confidence Interval 0.43%,1.00%). There were 68 studies reporting associations with hospital admissions for respiratory disease, with an overall effect estimate of 0.57% (0.33, 0.82) increase per 10 µg/m^3 increase in NO_2, and for cardiovascular hospital admissions the estimate was an increase of 0.66% (0.32, 1.01), supported by 52 studies. An additional systematic review and meta-analysis looked at associations of NO_2 and asthma emergency room visits or hospital admissions. Across 66 studies, they found a significant association, with a 10 µg/m^3 increase in NO_2 associated with a 1.8% increase in hospital attendance[409]. More recently, we published a paper that used causal

modeling methods to look at the association between daily NO_2 exposure and daily deaths in 105 U.S. cities. These included a substantial portion of the U.S. population and over 7 million deaths. We reported a significant association after controlling for the effects of $PM_{2.5}$, with a 2.6% increase (95% CI 1.8%, 3.4%) in daily deaths for each 10ppb increase in NO_2. This was larger than the previous meta-analysis[78].

These are too strong effects from too many studies to ignore. The World Health Organization agrees with this conclusion. In a report prior to the publication of the above meta-analysis, the HRAPIE project of WHO reported that a panel of experts recommended computing deaths and respiratory hospital admissions associated with increases in NO_2 concentrations[17].

There is support for this conclusion from other recent studies examining related outcomes. The Sisters Study looked at almost 44,000 women in the U.S. and reported that a 10-ppb increase in NO2 was associated with a 0.4-mmHg (95% CI: 0.2, 0.6; $p < 0.001$) higher pulse pressure[308]. Pulse pressue is the difference betweeen systolic and diastolic blood pressure, and increases in that difference are associated with heart disease. This finding makes an association with mortality more plausible.

In addition several recent studies have examined long-term exposure to NO_2 and mortality rates. In the CanCHEC cohort study, discussed in the ozone section, the investigators simultaneously examined the effects of

$PM_{2.5}$, ozone, and NO_2 on annual mortality rates[48]. For NO_2, they reported significant association, with an 8ppb increase in NO2 associated with a 4.5% increase in the annual mortality rate, after accounting for the effect of ozone and $PM_{2.5}$. A new analysis of the ACS study reported that also controlling for $PM_{2.5}$ and O3, reported an association of NO2 with mortality in that cohort[49]. These studies just add to the evidence reported in the systematic review of Faustini and coworkers[410]. They identified 23 papers between 2004 and 2013 that examined the association between long term exposure to NO_2 and long term mortality rates. They reported a 10 $\mu g/m^3$ increase in annual NO_2 concentrations was associated with a 4% increase in annual mortality.

Other types of evidence support a role of long-term NO2 exposure and the development of asthma, not just its exacerbation. For example, Lin and coworkers geocoded the residential addresses of children admitted to the hospital in Erie County New York (excluding Buffalo) for asthma, and age matched controls admitted for non-respiratory conditions[411]. These were linked to data on vehicle miles traveled on their street. The odds of asthma (adjusted for poverty level) for living within 200m of a street with the highest tertile of traffic density was 1.93 (95% CI 1.13, 3.29) and the asthmatic children were more likely to have truck traffic on their street. Trucks produce much more NO2 than cars. Another study analyzed data from two birth cohorts totaling 1,756 children in Munich[412]. Geographic Information System modeling was used to estimate the

concentrations of traffic related particles and NO_2 outside the birth addresses of all the children. These pollutants were associated with dry cough at night in the first year of life. Brauer and coworkers examined a birth cohort of approximately 4,000 children in the Netherlands, and found traffic related pollution was associated with increased risk of wheeze and physician diagnosed asthma[413]. A case-control study of 6147 children in Nottingham, England found increased risk of wheeze associated with living within 90 m of a roadway[414].

Also, prenatal exposure to nitrate particles, which are exclusively produced by NO2 in the atmosphere, were associated with greater odds of asthma in children in the ACCESS pregnancy cohort[415].

III.C.3. Mechanistic Studies

As noted in the 2016 ISA for NO_2,

> "The key evidence that short-term NO_2 exposure independently can trigger an asthma attack is the increased airway responsiveness and allergic inflammation induced by NO_2 exposure in controlled human exposures studies. ... Allergic inflammation and airway responsiveness are hallmarks of asthma attacks; thus, this evidence supports epidemiologic results, which consistently link short-term increases in ambient NO_2 concentration with increases in hospital admissions and emergency department visits for asthma, increases in respiratory

symptoms and airway inflammation in people with asthma, and decreases in lung function in children with asthma."

That is, when individuals are exposed to NO_2 in a laboratory setting, compared to clean air, they manifest increased inflammation in their lungs, and responsiveness, which is the term for the constriction of the breathing passages when stimulated. This is precisely what an asthma attack is.

At a more basic level, we know that after entering the lung, NO_2, which is an oxidizing compound, can contribute to the formation of reactive oxygen species in the lung. These in turn can oxidize the fatty acids that form the membrane of the epithelial cells lining the lung, which can change their permeability. They can also alter enzymes, proteins, etc. In particular, changes in lung antioxidant activity have been seen in NO_2 exposed animals.

As reported in the $PM_{2.5}$ section, telomeres are the end caps of chromosomes and are critical to reproducing the chromosomes correctly when cells divide. As such, shorter telomere length is a marker of increased risk of disease. A recent study looked at prenatal and childhood exposure to NO_2 and $PM_{2.5}$ with telomere length in white blood cells of children in six birth cohorts across Europe. Controlling for the effect of $PM_{2.5}$, an increase of prenatal NO2 of 14 $\mu g/m^3$ was associated with a −1.9% (−3.3%, −0.6%) shorter telomere length. This is consistent with NO2 being a gas producing oxidative stress, as telomeres are rich in elements sensitive to

oxidation. This is consistent with another recent study showing that distance to major road was a predictor of placental telomere length. Studies of exacerbation of COPD by NO2 have been mixed with some reporting no association[416, 417] and others reporting small increases, but only at high exposures [418, 419]. In contrast, there is substantial toxicological support for NO_2 increasing susceptibility and response to respiratory infections[420].

With respect to cardiovascular outcomes, controlled human exposure studies have not found increases in blood pressure or changes in cardiac output following NO_2 exposure[421, 422]. However, an acute exposure to NO_2 produced tachycardia in one occupational report. A controlled human exposure study reported an effect of 2-hour exposure to NO_2 on heart rate variability, cholesterol level, and that in combination with $PM_{2.5}$ exposure produced increased inflammatory markers in lung lining fluid not seen with either exposure alone (Huang et al., 2012b).

III.C.4. Conclusions

NO2 exposure is a cause of increased respiratory ailments, and very likely to be a source of increased mortality.

IV. QUANTITATIVE RISK ASSESSMENT

IV.A. Justification for Risk Assessment

International scientific consensus concurs that our knowledge about the causality and size of the effects of air pollution on increased deaths

allows us to quantify the number of deaths that may be avoided by reducing particles and ozone in the air. For example, the World Health Organization, as quoted in the beginning of this report, performed quantitative risk assessments based on the epidemiologic evidence for estimating the effects of particles in Europe. Subsequently, as part of its announcement of new standards, the WHO performed a world-wide estimate and quoted those numbers. The WHO particle standard and quantitative mortality estimates were based, as it noted, on expert consultation with over 80 scientists worldwide. WHO's most recent summary on the health effects on ambient air pollution, available online at http://www.who.int/airpollution/ambient/health-impacts/en/, states:

> "Ambient (outdoor air pollution) is a major cause of death and disease globally. The health effects range from increased hospital admissions and emergency room visits, to increased risk of premature death.
>
> An estimated 4.2 million premature deaths globally are linked to ambient air pollution, mainly from heart disease, stroke, chronic obstructive pulmonary disease, lung cancer, and acute respiratory infections in children."

WHO further explains that worldwide ambient air pollution accounts for:

- *25% of all deaths and disease from lung cancer*
- *17% of all deaths and disease from acute lower respiratory infection*

- *16% of all deaths from stroke*
- *15% of all deaths and disease from ischaemic heart disease [and]*
- *8% of all deaths and disease from chronic obstructive pulmonary disease*

Again, such quantification comes from doing a risk assessment.

Similarly, another group of scientists at WHO prepared the Global Burden of Disease estimate,[423] which estimates cases attributable to various diseases and exposures worldwide. In 2006, this group estimated that particulate air pollution produced 811,000 deaths per year in the world population that lived in cities (www.dcp2.org/pubs/gbd/4/Table/4/A107). They also quantify the benefits of reducing ozone concentrations. This estimate came from applying the epidemiology studies of long-term exposure and mortality rate, described previously in this report, to produce quantitative estimates of deaths due to particles and ozone. This approach has also been standardly used in the peer reviewed scientific literature for over a decade, including in the official journal of the Society for Risk Analysis.[424-431]

In its review in 2010 of the Policy Assessment for the revision of the $PM_{2.5}$ standard, the CASAC approved a document that makes use of the quantitative risk assessment in estimating the benefits of reducing $PM_{2.5}$ concentrations. The CASAC said:

> "For the fine particle standard, the *Policy Assessment* draws heavily on the analyses in the *Quantitative Health Risk Assessment for Particulate Matter*, which are based on the assumption of a linear no-threshold model for risk. The *Risk Assessment* covers the evidence supporting this model, drawing on the findings of the ISA, particularly the finding of associations of PM indicators with morbidity and mortality at concentrations measured in the United States."

CASAC also concluded:

> "CASAC concludes that the levels under consideration are supported by the epidemiological and toxicological evidence, as well as by the risk and air quality information compiled in the Integrated Science Assessment (December 2009), Quantitative Health Risk Assessment for Particulate Matter (June 2010) and summarized in the Second Draft Policy Assessment. Although there is increasing uncertainty at lower levels, there is no evidence of a threshold (i.e., a level below which there is no risk for adverse health effects)."

(EPA-CASAC-10-015, Letter dated Sept. 10, 2010, to the Administrator of EPA).

Recently, the United Nations Environment Program published a report estimating the benefits of air pollution control strategies that both reduced harmful pollutants and short-lived climate forcing compounds. That

assessment also underwent extensive scientific review, and estimated the mortality benefits of reducing particle concentrations using the mean of the EPA Expert Elicitation slopes.[90] That report concluded, "**Full implementation of the identified measures could avoid 2.4 million premature deaths (within a range of 0.7–4.6 million) and the loss of 52 million tonnes (within a range of 30–140 million tonnes), 1–4 percent, of the global production of maize, rice, soybean and wheat each year.**" (United Nations Environment Program report, at 3).

This report has been converted into a scientific journal article, and published in Science, perhaps the most distinguished peer-reviewed scientific journal in the world.[432]

Again, the U.S. National Academy of Sciences also endorsed the use of epidemiology to perform estimation of avoided early deaths in its report on benefit estimation and risk analysis for air pollution standards.[12] In that 2002 report, the Academy endorsed the use of concentration-response functions such as we used to estimate the benefits of incremental controls of particulate air pollution—precisely the case at hand.

The European Union, after its detailed evaluation of the scientific basis for particle standards set standards, and developed strategies to reduce particle levels. In EU Clean Air For Europe COM (2005) 446 Final Communication From The Commission To The Council And The European Parliament, it states that the goal of the strategy is a "47% reduction in loss of life expectancy as a result of exposure to particulate matter;

[t]o achieve these objectives, SO2 emissions will need to decrease by 82%." That is, it states the consensus that particles kill people, and that reducing particles will save lives, and by computing the amount of reduction in lost life expectancy that will occur following implementation of the strategy, it clearly indicates that such risk assessments are reasonable. Doing so, it goes on to state:

> "Concerning health impacts, currently in the EU there is a loss in statistical life expectancy of over 8 months due to $PM_{2.5}$ in air, equivalent to 3.6 million life years lost annually. The level of ambition chosen for this Strategy has been estimated to deliver at least €42 billion per annum in health benefits."

More recently, the Gobal Burden of Disease has been updated to use exposure estimates outside of large cities and estimate the worldwide impact of $PM_{2.5}$. Their latest estimate is that air pollution is responsible for 5.5 million early deaths per year, overwhelmingly from $PM_{2.5}$ [14] This year the Lancet Commission on Pollution and Health confirmed those findings.

Hence, the scientific consensus is not merely that the concentration response functions used to estimate quantitative risks are reasonable, but multiple national and international organizations have in fact used them. Similarly, EPA has performed quatitative risk assessments for ozone and NO2 health effects in the RIAs for those pollutants, and again, this is similar to efforts by the Global Burden of Disease and WHO.

Moreover, this is also like the quantitative risk assessments routinely done by public health agencies for other exposures. Indeed, the Centers for Disease Control (CDC) and the AHA do just that every year. Consider the "risk factor" of smoking. About it, CDC states that "[s]moking leads to disease and disability and harms nearly every organ of the body" and that "[c]igarette smoking is responsible for more than 480,000 deaths per year in the United States" (https://www.cdc.gov/tobacco/data_statistics/fact_sheets/fast_facts/index.htm). Cigarette smoking is not listed as a cause of death on any death certificate, and more of these 480,000 deaths come from heart disease than from lung cancer. Similarly, regarding the "risk factor" of high blood pressure, the CDC says that "*[h]igh blood pressure was a primary or contributing cause of death for more than 410,000 Americans in 2014*" (https://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_bloodpressure.htm).

CDC also considers $PM_{2.5}$ as a risk factor for which it can calculate attributable deaths. Regarding $PM_{2.5}$ as a "risk factor," the CDC says:

> "Changes observed in people exposed to $PM_{2.5}$ include: increased airway inflammation and sensitivity, decreased lung function, changes in heart rhythm and blood flow, increased blood pressure, increases in the tendency to form blood clots, and biological markers of inflammation. These health effects cause increases in symptoms, emergency department visits, hospital admissions, and deaths from

heart and lung diseases."

(https://ephtracking.cdc.gov/showIndicatorPages.action?selectedContentAreaAbbreviation=11&selectedIndicatorId=75&selectedMeasureId=.) The CDC further goes on to quantify those effects, stating that "*[f]or example, according to 2009 data available on the Tracking Network, a 10% reduction in $PM_{2.5}$ could prevent:*

- *more than 400 deaths per year in a highly populated county, like Los Angeles County;*
- *about 1,500 deaths every year in California; and*
- *over 13,000 deaths across the nation."*

(https://ephtracking.cdc.gov/showAirHIA.action.)

IV.B. The Fundamentals of a Risk Assessment

Conducting a risk assessment for an action, such as excess emissions of NOx, has several fundamental components. First, given estimates of how many tons of increased emissions occured, one must convert that into estimates of the change in concentrations in the air, at the locations where the population for whom the risk assessment is being conducted live. This is often done using chemical transport models, as was done in the RIAs for the NAAQS. These models take as inputs EPA's emission inventories, describing the locations and amounts of emissions, including from natural sources as well as anthropogenic sources. These are combined with detailed weather data from the National Atmospheric and Oceanic

Administration, which include factors such as wind patterns and weather fronts, temperature, etc. at mutiple altitudes, which allow the model to estimate where the emissions go. Finally, the models contain complex, nonlinear chemistry to capture the reaction of the various chemicals in the air, such as NOx, which result in formation of particles and ozone. These models are run twice, with and without the excess emissions, and the differences are the incremental exposures due to the excess emissions. Typically estimates are summarized by dividing the country into geographic units, such as counties, because other parts of the health impact assessment are only available at those levels.

Second, given the estimated differences in concentrations in each county, we need to estimate the difference (between excess exposure and no excess exposure scenarios) in mortality or in morbid events in that county. The mortality-pollutant studies provide estimates (slopes) that basically relate change in pollution concentration to a percent change in the mortality rate. Therefore, if we multiply the change in the pollution concentration in a county (resulting from the emissions increase) by the slope from the mortality studies, we get an estimate of the percent change in the mortality **rate** in that county.

However, we want to know how many actual deaths occured, not how much the **rate** changed. This requires two more steps. First, we convert the percent change in mortality rate into an absolute change in the mortality rate. To do this we need to know the baseline mortality rate. Suppose, for

example, we had estimated that the difference in exposure between the two scenarios resulted in a 1% change in the mortality rate. Multiplying this times the baseline mortality rate (for example 8 deaths per 1000 population) would give the change in the absolute mortality rate attributable to that difference, which in the above example would be 1% of 8, or 0.08 deaths per 1000 persons living in the county. Finally we need to multiply by the population of the county to get the estimated change in deaths per year in the county. Mathematically, the formula is:

$$Deaths = Pop * Y_0 * (1-exp^{-\beta \Delta X}) \quad (1)$$

Where Pop is the population of the county, Y_0 is the baseline mortality rate in the county, β is the slope of the pollution mortality relation, and ΔX is the change in pollutant concentrations. We repeat this process for every county in the U.S. that we study to get the estimated total change in deaths as a result of the increased concentration of that pollutant due to the increase in emissions. This process is repeated for each type of event (e.g., deaths, heart attacks, hospital admissions). Running chemical transport models is expensive and time consuming, and in a case such as this, lack of knowledge of exactly where the excess emissions from Navistar engines occurred makes it unfeasible. EPA has also developed reduced form methods that are used in such a case. For example, in the RIA for the repeal of the clean power plan, EPA used a benefits per ton approach. This approach still relies on a chemical transport model. However, it fits the chemical transport model for a reference case of

emissions, and computes the health effects, and associated monetary costs, from those emissions. Those can be divided by the number of tons of emissions to derive a benefits per ton from emissions reduction, or equivalently, cost per ton from emissions. More recently, Wolfe and coworkers used a source specific model chemical transport model to derive the costs per ton of emissions from multiple categories of mobile sources[433]. These costs were restricted to those due to the particles formed by the emissions, and excluded ozone, or direct NO2 health effects. They report those costs per ton of NOx emissions from heavy duty Diesel as $14,000. That is, on average, the same type of engines as the Navistar engines in this case produce $14,000 in economic damage per ton of emissions. Since the Navistar engines were sold throughout the country and subsequently driven widely over the United States,[440] this national average approach to estimating costs per ton seems appropriate to this case. To derive that cost, Wolfe and coworkers estimated deaths per ton and converted the number of deaths into monetary values, as discussed below.

EPA, in its revised RIA for the Cross State Air Pollution Rule, accounting for the ozone standard, adopted a similar benefit per to approach to estimate the health and monetary benefits of the reduced ozone consequent to reducing a ton of NOx. They report (p134) those benefits range from $6,000 to $9,900 without discounting and from $1,200 to $2,500 with discounting[434]. These numbers assume that there is no effect of long-term ozone exposure on mortality rates.

Finally, a benefit analysis seeks to convert these numbers of events into monetary terms so that they can be compared to the costs of controls. I have also conducted this analysis. Below, I first describe the sources of the components of these estimates, and then I subsequently report the results.

IV.B.1. Valuation

In order to estimate the damages due to emissions of NOx, the increased health effects estimated above are converted into monetary terms. This is done for two reasons. First, when comparing policy alternatives, it is often useful to compare the benefits of emission reductions to the costs of providing those reductions. Costs, of course, are computed in dollars, so it is advantageous that benefits be monetized as well. This benefit-cost approach to public policy evaluation is used extensively by federal agencies[435-437].

The second reason that benefits are monetized is that air pollution has many different impacts on society. These include premature death, increased rates of illness, and increased hospital admissions. Valuation provides a means to express these impacts in a common unit. This facilitates aggregation of impacts across endpoints. For the purposes of this analysis, we will model mortality and morbidity impacts. The discussion of valuation methodology follows accordingly.

How do people value changes to mortality risks?

Translating mortality impacts into monetary damages relies on statistical techniques to estimate how people and societies value mortality risks. This estimate is not a price on a life. It is obtained by estimating how much people are willing to pay for small reductions in their risk of death. This is appropriate because we can tell people that lowering $PM_{2.5}$ concentrations will lower their risk of dying, but we cannot identify which people are the ones who will (or will not) die. Hence the quantity we want to value is risk reduction. If people are willing to pay, for example $100 for a reduction in risk of 1 in 10,000, then if 10,000 people purchased an intervention that reduced their risk by 1 in 10,000, there would, on average, be 1 fewer death in the population, at a cost of 1,000,000 dollars. To ease computation of benefits, instead of estimating the increased risk to each person in the U.S. and multiplying by the value to them of avoiding that risk ($100 in the above example), it is more convenient (but identical) to estimate the number of excess deaths and multiply that (in the above example) by $1 million. The latter number is referred to as the value of a statistical life in the economics literature[438]. This estimate is based on research where people are asked how much they would pay for consumer products (such as water filters) that reduce risk. Alternatively, there are studies that examined how much more employers have to pay employees (adjusting for age, education, and

experience, etc.) to compensate for taking an increased risk of accidental death.

Formally the VSL is defined as the maximum rate at which an individual would pay to reduce their chance of death by a small amount in a certain time period (often the current year), or alternatively, the minimum compensation that an individual would require in order to accept an increase in the risk of death in a specified period. Although the preceding discussion of the VSL concept is abstract, instances of people actually making such a tradeoff (between money and mortality risks) are evident in market behavior. A typical example is purchasing equipment or devices that reduce the risk of death such as water filters or smoke detectors. People also show a willingness to exchange money for mortality risks in the workplace.

Broadly speaking, these two approaches are used to estimate VSLs, called revealed preference methods and stated preference methods in the economics literature. Revealed preference methods have used regressions to measure the effect that occupational mortality risk has on wages across a wide range of occupations and, therefore, risk levels[438], the second type of study described above. These studies carefully control for many characteristics of the worker and the occupation in order to estimate the risk-wage tradeoff free of confounding.

In contrast to revealed-preference techniques which focus on choices that individuals make in market transactions, stated preference studies

ask survey respondents how, in hypothetical situations, they would make tradeoffs between money and mortality risk. Stated preference studies have been used to elicit respondent's mortality-risk money tradeoff in various contexts including: traffic-related risks, health-related risks, and risks of death in the workplace[439].

The findings of either the revealed preference or the stated preference studies reflect an average rate of tradeoff between money and the chance of death. EPA has reviewed the VSL literature most recently in the Regulatory Impact Assessment for the $PM_{2.5}$ Ambient Air Quality Standards[102].

IV.B.2. Choosing the Dose-Response slopes

A key issue in a risk assessment is the choice of slopes relating the various exposures to health endpoints. This was reviewed extensively by EPA in their most recent Regulatory Impact Analysis.

The most important slope for this estimation is the one with mortality. EPA presents two slopes from two studies, and carries that through into their estimate of benefits per ton. There are several reasons I do not accept this approach. First, this ignores all of the other cohort studies (over 50 of them) on $PM_{2.5}$ and mortality, many more recent and with better exposure measures, which is hard to justify. Second, most studies seem to be distributed around a central tendency, which argues for choosing a central tendency. And third, many recent studies have been conducted at the lower concentrations of air pollution that now prevail.

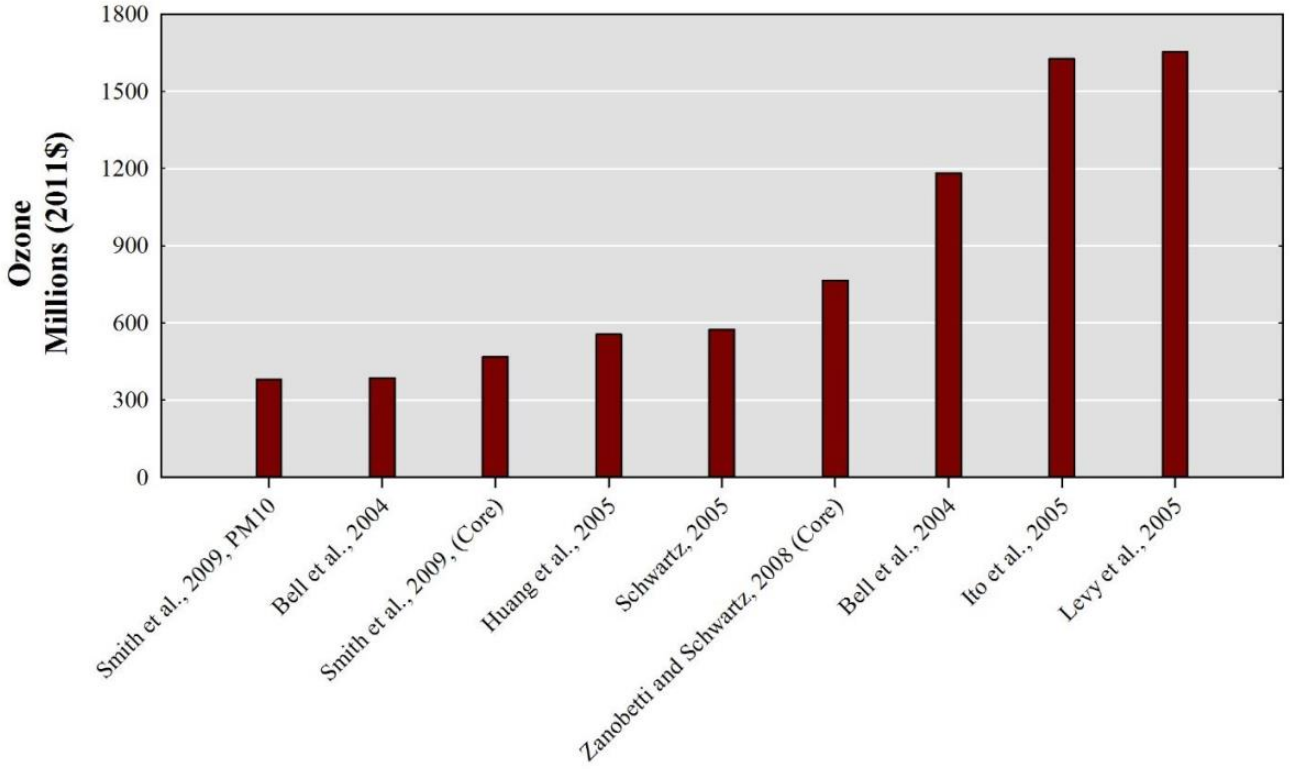
In a recent paper, we examined 135 estimates of the C-R slope from 53 different cohort studies, and fit a meta-analysis that combined the estimates, and also showed how they varied with concentration.[26]. We systematically searched all published cohort studies that examined the association between long term exposure to $PM_{2.5}$ and mortality. This systematic review and meta-analysis was done in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement 44. We identified studies through a search in Pubmed (Medline Ovid), Embase, EBSCO, Web of Science and Global Health on CAB databases (last accessed on April 20, 2017).

A meta-analysis is essentially a weighted average of the C-R slopes from all the studies, giving more weight to those studies that have tighter confidence intervals. In addition, we examined characteristics of each study that might influence the C-R slope, including the $PM_{2.5}$ concentrations in the study areas. A key additional feature of a meta-analysis is that it directly accounts for heterogeneity in C-R slopes across different studies. It uses the confidence intervals within each study to estimate how much variation in slopes one would expect just by random chance, and uses the remaining variation in the slopes between studies as an estimate of the additional variation due to differences between studies. Our confidence intervals for the central estimate from the meta-analysis incorporate this additional uncertainty. Importantly, 14 studies were conducted on cohorts with mean exposure less than 10 $\mu g/m^3$,

giving us considerable information on which to estimate the C-R slope at lower exposures which now prevail in the United States.

Using those studies, we found a nonlinear C-R slope, with lower slopes at higher concentrations no longer seen in the U.S. At a concentration of 10 $\mu g/m^3$, we found a 1 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 1.29% increase in all-age all-cause mortality (95% CI 1.09, 1.50). Since 10 $\mu g/m^3$ is approximately the mean $PM_{2.5}$ concentration in the U.S. this seems the most appropriate choice. It is almost identical to the results in the Wolfe paper calculating benefits per ton using the $PM_{2.5}$-mortality coefficient from the Lepeule paper[30], so I have used those results from Wolfe based on the Lepeule coefficients as the central estimate of the $PM_{2.5}$ related benefits per ton. This results in a benefit per ton of NOx reduction of $14,000. The Wolfe et al. paper does not give confidence limits for this prediction, so we have used the confidence intervals from the Lepeule paper on which it was based, providing a 95% confidence interval of ($7,000, $21,000).

The EPA derived benefits per ton of NOx through its impact on ozone formation using dose-response estimates between ozone and mortality from two papers. The paper with the higher estimate has an effect size that is lower than those from meta-analyses of the ozone literature (Levy 2005, Ito 2005), as indicated by the below figure from the EPA RIA.



Given this, I have taken the Zanobetti and Schwartz estimate as a midpoint and not high-end number. Using that, the benefits per ton of NOx reduction through its role in ozone formation are $2,500 using discounting. Using the confidence interval from the Zanobetti and Schwartz paper provides a confidence interval for the benefits per ton of ($1,665, $3,333).

IV.B.3. Long term ozone exposure and Mortality

The issue of long-term effects of ozone on mortality is one where I have used new information to update the concentration response slope in the ozone RIA. In general, studies of long-term exposure report large concentration-response slopes than studies of short-term exposure, and are

assumed to encompass the latter. There are 6 studies that have reported concentration-response slopes between long-term ozone exposure and chronic mortality rates. Two of them examined sensitive populations, and, as expected, reported larger slopes than the rest, which are not easy to extrapolate to the general population. The remaining four are the studies of Jerrett[381], of Turner[49], and of Crouse[48] and Di[385]. Since the Turner study is an extended follow-up of the same cohort used by Jerrett, I have averaged the dose-response slopes from the remaining three studies. For all-cause mortality, the Crouse study reports that after controlling for $PM_{2.5}$ and NO2, a 9.5 ppb increase in ozone was associated with a 1.8% increase in deaths from all cause (95% CI 1.0%, 2.6%). The Turner study reported that a 10 ppb increase in ozone was associated with a 2% (95% CI 1%, 4%) increase in deaths from all cause, controlling for $PM_{2.5}$ and NO_2. The Di study reported that 10ppb increase in ozone was associated with a 1.1% (95% CI, 1.0 to 1.2) increase in deaths from all cause. Based on this, I computed the health impact of the increased ozone due to Navistar's excess NOx emissions assuming a slope of 1.6% increase in all cause deaths for each 10 ppb increase in annual average ozone. Because there are fewer ozone studies than $PM_{2.5}$ studies, and the mechanistic data is not quite as strong, I have computed these effects as a sensitivity analysis. This then needs to be adapted to the benefits per ton approach. The long-term percent change in deaths per 10ppb change in ozone above is approximately 3 times the estimate for short-term effects in the Zanobetti

and Schwartz paper used in the CSAPR RIA. I have assumed that the acute effects are captured in the chronic exposure effects, and therefore, in the sensitivity analysis, I have assumed the ozone-related benefits are 3 times what I have used in my base case analysis, or $7,500 per ton of NOx.

IV.B.4. Results of the Risk Assessment.

Based on the information provided to me by ERG,[441-443] I computed the health and economic damages under the three different scenarios for the Subject Engines: excess emissions of the Subject Engines relative to not being sold; excess emissions of the Subject Engines relative to the 0.2 standard, and excess emissions of the Subject Enines relative to a 0.5 NOx FEL. For the Credit Engines, there is only the one value for the excess emissions. The harm form the Credit Engines is added to the harm from the Subject Engines in each scenario. The results are shown in the table below.

Excess Emissions from the Subject Engines Relative to not being sold			
	Tons	Resultant Deaths and 95% CI	Economic Damage (billions of Dollars) and 95% CI
Past Emissions	(b) (4) [redacted]	[redacted]	[redacted]

Future Emissions	(b) (4)
Credit Emissions (past)	
Credit Emissions (Future)	
Total Emissions	
Total Emissions including Chronic Ozone Effects	

Excess Emissions of the Subject Engines Relative to the 0.2 Standard

Past Emissions	(b) (4)
Future Emissions	
Credit Emissions (past)	
Credit Emissions (Future)	
Total Emissions	

Total Emissions including Chronic Ozone Effects	(b) (4)
Excess Emissions of the Subject Engines Relative to a 0.5 FEL	
Past Emissions	(b) (4)
Future Emissions	
Credit Emissions (past)	
Credit Emissions (Future)	
Total Emissions	
Total Emissions including Chronic Ozone Effects	

Based on the discussion above, the benefits of reducing NOx emissions through their impact on $PM_{2.5}$ concentrations are $14,000 per ton, and through their impact on ozone formation are $2,500 per ton, for a total of $16,500 per ton (95% CI $23,550, $9,450). To estimate confidence intervals for these costs, I have used the standard formula for the variance of a

sum of two independent parameters (Var(A+B)=Var(A) +Var(B)). Hence, the health costs imposed on the U.S. population by the Navistar excess emissions are (b) (4) [redacted]

If we take into account the likely long-term effects of ozone exposure, then the central estimates of the costs imposed rise to (b) (4) [redacted] This does not include the possible effects of short-term NO_2 exposure on mortality, which would add to these amounts.

V. CONCLUSIONS

In summary, NOx emissions produce particles and ozone, which, in turn, produce increased numbers of deaths, heart attacks, hospitalizations for pneumonia, asthma, and other serious health impairments. There is no evidence for a threshold for these effects, which means that any incremental exposure is associated with incremental deaths, heart attacks, etc. in the general population, and the economic costs of those effects is substantial. In addition, there are health effects of NO_2 itself.

Appendix A. Medical Societies Letter on Ozone

American Academy of Pediatrics • American College of Preventative Medicine American Heart Association • American Lung Association American Medical Association • American Public Health Association • American Thoracic Society • Asthma and Allergy Foundation of America Children's Environmental Health Network National Association of County and City Health Officials National Association for Medical Direction of Respiratory Care • Health Care Without Harm • Trust for America's Health

Re: EPA Docket I.D. No: EPA-HQ-OAR-2008-0699

Dear Administrator McCarthy:

As national organizations representing medical societies, public health and patient advocacy organizations, we write to provide comments to the U. S. Environmental Protection Agency on the proposed Ozone National Ambient Air Quality Standards. Our organizations appreciate and would like to express our support to the EPA for moving forward to update the current ozone standard, and welcome this opportunity to provide input to this process, which we hope will result in a standard that is better protective of public health. Our organizations urge you to select a level for the primary health standard that will meet the Clean Air Act requirement to protect the health of the public with an adequate margin of safety: 60 parts per billion (ppb).

EPA Must Protect the Health of the Public, including Sensitive Populations

The Clean Air Act establishes the primary National Ambient Air Quality Standard to protect public health from the nation's most widespread air pollutants. The Clean Air Act directs the Administrator to set standards that are "requisite to protect public health" with "an adequate margin of safety" (42 U.S.C. § 7409 (b) (1)). The list of populations who risk demonstrated harm from ozone pollution has grown significantly from the previous review. Children, people with asthma and other lung diseases, seniors, outdoor workers and people who have low socioeconomic status have long been shown to be vulnerable to ozone. Newer evidence shows some otherwise healthy adults are especially sensitive to ozone exposure because of limitations in some nutrients and certain genetic variants. In addition to these groups, the EPA's Integrated Science Assessment has documented evidence that suggests increased risk to fetal development and to cardiovascular harm (EPA, Integrated Science Assessment, 2013). Health-based standards must be set at levels that will protect all people, but particularly these sensitive groups. Ozone poses a grave threat to public health at levels well below the current standard. The current standard of 75 ppb fails to meet the requirements of the Clean Air Act. Clinical and epidemiological studies have repeatedly shown that breathing ozone can threaten life and health at concentrations far lower than the 75 ppb 8-hour average standard. Extensive, public reviews of the large body of evidence by EPA's independent science advisors, the Clean Air Scientific Advisory Committee (CASAC), and by EPA staff scientists have confirmed

that the 2008 primary ozone standard is set at a level that is too weak to protect public health. In fact, three successive CASAC panels -- each under different leadership -- have reached the same conclusion: the 2008 standard should not be retained.

- As part of the advice to the EPA during the previous review that ended in 2008, CASAC sent letters repeatedly supporting a standard between 60 and 70 ppb (Henderson, 2006; Henderson, 2007). After EPA published its final decision in 2008, CASAC sent a rare letter to the Administrator commenting on the decision. The CASAC stated unequivocally that they disagreed with the decision to set the standard at 75 ppb. These scientists notified the Administrator that they "do not endorse the new primary ozone standard as being sufficiently protective of public health." (Emphasis in the original.) They urged that the Administrator or his successor "select a more health-protective" standard in the next review cycle (Henderson, 2008). It is important to note that their decision was based on the scientific evidence as it stood in 2006, the close of that review period.

- When asked to reevaluate the evidence during EPA's ill-fated reconsideration of the 2008 standard in February 2010, CASAC again was explicit: "EPA has recognized the large body of data and risk analyses demonstrating that retention of the current standard would leave large numbers of individuals at risk for respiratory effects and/or other significant health consequences including asthma exacerbations, emergency room visits, hospital admissions and mortality" (Samet, 2010).

- Now, the current CASAC has echoed this consensus again. In their letter to EPA on June 14, 2014, they stated it simply: "The CASAC finds scientific justification that current evidence and the results of the exposure and risk assessment call into question the adequacy of the current standard" (Frey, 2014).

We share the conclusion repeatedly presented to EPA by the CASAC: EPA cannot justify retention of the current standard based on the health evidence. Multiple CASAC reviews have recommended a standard between 60 and 70 ppb. Not only have the three separate CASAC committees, under three different Chairs, unanimously confirmed that the current ozone standard is not protective of public health, but each recommended that the standard should be set in the range of 60 to 70 ppb.

In each of the three comment letters the CASAC wrote to EPA Administrator Stephen L. Johnson, the independent experts charged with advising EPA unanimously recommended selection of an 8-hour average ozone NAAQS within the range of 60 to 70 ppb (Henderson 2006; Henderson 2007; Henderson 2008). During the reconsideration of the 2008 Ozone NAAQS, CASAC reaffirmed its support for the selection of an 8-hour average ozone NAAQS within the 60 – 70 ppb range (Samet, 2010). Again, that recommendation came based solely on the studies that had been available during the prior review, a period that closed in 2006.

Now able to fully consider the additional studies available in the 2007 to 2012 period, the most recent CASAC summarized extensive scientific

evidence in their recommendations to EPA for a range from 70 to 60 ppb: "The CASAC further concludes that there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb. The CASAC reached this conclusion based on the scientific evidence from clinical studies, epidemiologic studies, and animal toxicology studies, as summarized in the Integrated Science Assessment (ISA), the findings from the exposure and risk assessments as summarized in the HREA, and the interpretation of the implications of these sources of information as given in the Second Draft PA" (Frey, 2014). However, the CASAC concluded that new evidence showed that even that range is too broad, noting that "based on the scientific evidence, a level of 70 ppb provides little margin of safety for the protection of public health particularly for sensitive subpopulations" (Frey, 2014). "At 70 ppb, there is substantial scientific evidence of adverse effects as detailed in the charge question responses, including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation. Although a level of 70 ppb is more protective of public health than the current standard, it may not meet the statutory requirement to protect public health with an adequate margin of safety" (Frey, 2014). CASAC concluded the evidence showed that a level of "60 ppb would certainly provide more public health protection than a standard of 65 or 70 ppb and would provide an adequate margin of safety" (Frey, 2014). The significantly stronger scientific and medical evidence available in this current review led CASAC

to provide even more explicit comments than during the 2008 review and the subsequent reconsideration process. Their explicit conclusion that 60 ppb meets the requirement to provide more protection and an adequate margin of safety raises questions about EPA's decision to exclude 60 ppb from the proposal. Our organizations offer evidence that demonstrates why 60 ppb should be adopted as the level of the health-based standard. The evidence for a standard of 60 ppb has grown. The scientific and medical understanding of the mechanisms by which exposure to ambient ozone pollution harms human health has grown considerably stronger since 2007. The EPA evaluated 1,000 new studies in the current review, studies that have been published since the completion of the 2006 Criteria Document. These studies inform our understanding of the health impacts of ozone at low concentrations. Multiple chamber studies provide robust evidence of harm to healthy adults down to 60 ppb. Adding to previous research by Adams (2002) and Adams (2006), both Brown et al (2008) and Kim et al (2011) provide still more evidence that exposures down to 60 ppb can reduce lung function and cause inflammation that meets the American Thoracic Society's criteria for judging adversity. The subjects in these chamber studies were healthy young adults -- not children, the elderly, or people with asthma who are more susceptible to ozone. The chamber studies establish solid evidence that concentrations above 60 ppb would provide significant risk not only to many healthy adults, but most critically, to susceptible populations, including children, seniors and people with

asthma and other chronic lung diseases. Epidemiological studies provide real-world evidence for the need for 60 ppb. The analysis presented in the Policy Assessment digs deeper into six epidemiological studies in the U.S. and Canada and provides further real-world evidence that a standard of either 70 ppb or 65 ppb fails to provide adequate protection. These studies (Bell et al., 2006; Cakmak et al., 2006b; Dales et al., 2006; Katsouyanni et al., 2009, Mar and Koenig, 2009; Stieb et al, 2009) examined the positive and statistically significant associations from the most serious health threat—premature death—as well as from hospital admissions and emergency department visits. In most locations where increased risk was found, the ozone levels would have met the weaker standards of either 70 or 65 ppb, but would have failed to meet a standard set at 60 ppb. (Policy Assessment, pp. 4-13 to 4-15). A standard of 60 ppb would result in a far greater reduction in premature morbidity and mortality. The EPA's estimates show that compared to meeting a standard of 65 ppb or 70 ppb, meeting a standard of 60 ppb would prevent many more premature deaths and hospital admissions, asthma attacks and days missed at work and school. Looking just at the parts of the nation expected to meet a standard of 60 ppb by 2025 (not including California), EPA provides a table of these estimates based on established modeling projections. Nationwide Benefits of Attaining Standard in 2025 Throughout the United States (except California) Measure 60 ppb 65 ppb 70 ppb Premature Deaths Avoided in 2025 7,900 4,300 1,440 Asthma Attacks Avoided in Children in

2025 1,800,000 960,000 320,000 Respiratory Hospital Admissions Avoided in 2025 2,900 1,500 510 Asthma Emergency Department Visits Avoided in 2025 4,100 2,300 1,400 Missed School Days Avoided in 2025 1,900,000 1,000,000 330,000 Taken from Table ES -11 of the U.S. EPA, Regulatory Impact Analysis of the Proposed Revision to the National Ambient Air Quality Standards for Ground-level Ozone, November 2014. EPA -452/P-14-006.

Estimates based on modeling and assumptions explained in detail in the document. California was excluded because it is not expected to meet these standards in 2025. In 2025, the reduction in premature deaths expected with a standard of 60 ppb is projected to be nearly double that of a standard set at 65 ppb and more than five times the benefit of a standard set at 70 ppb. Growing evidence expands health effects of ozone exposure. Your decision must be founded in the strongest requirement of the Clean Air Act: that the NAAQS not only protect public health, but include an adequate margin of safety. In both the prior review ending in 2008 and in the 2010 reconsideration, our organizations recommended strongly that the primary 8-hour standard should be 60 ppb based on the available evidence. In addition to the strong evidence of increased morbidity from ozone down to 60 ppb, multiple well-reviewed studies had identified a new, strong association with premature death, with no discernable threshold, that made the risks to the large, vulnerable groups even graver. Even during the prior reviews, the evidence demonstrated that standards between 65 and 70 ppb would not be effective in protecting public health with an adequate

margin of safety. Since the 2008 standard, new research has added weight to the evidence showing the extensive impact of ozone. Research not only confirms the previous conclusions about ozone's impact on human health, but adds to and clarifies the impact on multiple physiologic systems, including respiratory and cardiovascular. Examination of long-term exposure has identified outcomes beyond the traditional concerns to include the central nervous system and reproductive and developmental effects. The growing evidence of effects associated with breathing ozone for longer periods adds to the urgency to set the most protective standard now to reduce those exposures. Comments to Docket ID NO. EPA-HQ-OAR-2008-069 6 Respiratory Health Effects, including Premature Mortality The largest body of research documents the impact of ozone on respiratory symptoms, lung function changes, emergency department visits for respiratory disease, and hospital admissions. Since the previous review large studies examining exposures in multiple cities and continents have shown the consistent and pervasive threats to respiratory health. New studies confirm the impact on children with asthma. Multiple studies demonstrated increased pulmonary inflammation (Berhane et al., 2011; Khatri et al., 2009; Barraza-Villerreal et al, 2008), and increased risk of hospital admissions (Silverman and Ito, 2010; Strickland et al., 2010). Several large studies looking at single cities and multiple cities confirm that breathing ozone increases the risk of hospital admission and emergency department visits for respiratory conditions (Katsouyanni et al,

2009; Lin et al., 2008a; Wong et al., 2009; Darrow et al., 2011); Stieb et al., 2009). Multiple- and single-city studies showed increased risk of respiratory hospital admissions and emergency department visits in cities that met the current ozone standard of 75 ppb (Cakmak et al., 2006; Dales et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009) or where most cities would have met standards set at either 65ppb or 70 ppb (Cakmak et al., 2006; Katsouyanni et al., 2009; Stieb et al. 2009). The American Thoracic Society summarized some of the new studies in the attached editorial in the American Journal of Respiratory and Critical Care Medicine advocating EPA adoption of a standard of 60 ppb (Rice, et al., 2015). 8-hour "Highlights of this new body of evidence include a study of emergency department visits among children aged 0 to 4 in Atlanta, which found that each 30 ppb increase in the 3-day average of ozone was associated with an 8% higher risk of pneumonia and a 4% higher risk for upper respiratory infection(5)[Darrow et al 2014]. Several studies have demonstrated dose-response relationships between ozone exposure and childhood asthma admissions at exposure levels in the 60 to 80 ppb range (6–9)[Strickland et al 2014, Strickland et al 2010, Gleason et al 2014, Silverman et al 2010]. Similar associations have been found for adult admissions for asthma(9– 11) [Silverman and Ito 2010, Glad et al 2012, Meg et al 2010] and COPD(12, 13)[Ko and Hui 2012, Media-Ramon et al 20076]. A population-based cohort study of generally healthy adults found that FEV1 was 56 mL lower after days when ambient ozone ranged from 59 to 75 ppb

compared to days with levels under 59 ppb(14) [Rice et al 2013]. Controlled human exposure studies have re-affirmed lung function decrements in healthy adults after exposure to 60 to 70 ppb of ozone (15,16) [Schelegle et al 2009, Kim et al 2011]. Perhaps of greatest concern, there is now stronger evidence of increased mortality in association with ozone (17–19)[Peng et al 2013, Romieu et al 2012,Zanobetti and Schwartz 2008], particularly among the elderly and those with chronic disease(20, 21)[Medina-Ramon and Schwartz 2008, Zanobetti and Schwartz 2011]". Cardiovascular Health Effects, including Premature Mortality Evidence is accumulating about the cardiovascular effects of ozone, with the strongest evidence for increased risk of premature death. Previous studies have shown adverse associations between ozone exposure and various cardiovascular health endpoints, including alterations in heart rate variability in older adults (Park et al., 2005), cardiac arrhythmias (Rich et al., 2006), strokes, (Henrotin et al., 2007) Comments to Docket ID NO. EPA-HQ-OAR-2008-069 7 heart attacks (Ruidavets et al., 2005), and hospital admissions or cardiovascular diseases (Koken et al., 2003). Newer large epidemiologic studies from the U.S. (Zanobetti and Schwartz, 2008b), Europe (Samoli et al., 2009) and Asia (Wong et al 2010) have provided evidence of premature death from cardiovascular effects, including two large studies that confirmed the effect after controlling for particulate matter exposure (Katsouyanni et al 2009; Stafoggia, 2010). Reproduction and Development Effects A growing body of

research raises concerns about longer-term exposure to ozone, particularly during pregnancy. Some toxicological studies warn that ozone may affect development of the pulmonary system and central nervous system. Several large studies in California and Australia point to association of prenatal ozone exposure with low birth weight and impaired fetal growth (Salem et al., 2005; Morello-Frosch, et al. 2010; Hansen et al 2007, Hansen et al 2008;Mannes et al 2005). Low birth weight is linked to increased risk of chronic disease as adults (Rogers et al., 2012; Berends et al., 2012). Central Nervous System Effects Increased research since the last review has expanded evidence of the potential effects on the central nervous system. Toxicological studies provide evidence that short- or long-term exposure to ozone may affect cognitive abilities, such as memory (Rivas-Arancibia et al., 1998), and may produce changes similar to those seen in human neurodegenerative disorders (Rivas-Arancibia et al., 2010; SantiagoLópez et al., 2010; Guevara-Guzman et al., 2009). The only human epidemiological study found an association for long-term ozone exposure with reduced performance on specific tests (Chen and Schwartz 2009). While more research is clearly needed, these studies provide added weight for selecting the most protective level. Mortality Effects Breathing ozone can kill. Short-term increases in ozone were found to increase deaths from cardiovascular and respiratory causes in a large 14-year study in 95 U.S. cities. The relationship between mortality and ozone was evident even on days when pollution levels above 60 ppb were excluded from the analysis.

(Bell, et al., 2004). A series of meta-analyses and multi-city studies has documented an increase in premature death following ozone exposures below 75 ppb, particularly among the elderly (Bell, et al., 2005; Levy et al., 2005; Ito et al., 2005). Furthermore, research has focused on controlling for weather variables in assessing the effect of ozone on mortality. A case crossover study (Schwartz, 2005) of more than one million deaths in 14 U.S. cities found that "the association between ozone and mortality risk is unlikely to be confounded by temperature." Multiple new studies have confirmed that ozone causes premature deaths (Zanobetti and Schwartz, 2008b; Samoli et al., 2009; Wong et al 2010) and provided evidence that these deaths occur even after controlling for other pollutants, including particulate matter (Stafoggia, 2010; Katsouyanni et al., 2009). Of special concern the risk of premature death from ozone showed up more frequently in communities with higher unemployment or that had a higher percentage of Black/African-American population, as well as in individuals who were Black/African-American or who had lower socioeconomic status. (Median-Ramón and Schwartz, 2008). EPA needs to ensure the strongest, most protective standards are in place to prevent this deadly pollutant from threatening the lives of thousands of Americans. Comments to Docket ID NO. EPA-HQ-OAR-2008-069 8 Millions of Americans face greater risk from breathing ozone pollution Research has shown that many groups face greater risk from breathing ozone pollution or are more vulnerable to the harm because of their activities or residence. Their greater risk may come from

age, preexisting diseases or genetics, as well as income. Greater vulnerability may stem from outdoor occupations or activities or from living in areas with higher ozone exposures. Children and adolescents Children are acutely vulnerable to the hazardous effects of air pollution(AAP, 2004). Relative to adults, children tend to spend more time out of doors, they are often more physically active, they breathe more rapidly, their airways are narrower and they inhale relatively more pollutants in proportion to their body weight (AAP, 2003). Additionally, lung growth continues long after birth, with as much as 80 percent of the alveoli developing during childhood and adolescence (Diertert et al., 2000). Epidemiologic evidence indicates that children face additional health risks beyond the adverse effects observed in the general population. Children experience acute effects such as difficulty breathing (Triche et al., 2006), increased hospitalizations (Burnett et al., 2001), and emergency room visits (Tolbert et al., 2000) from ozone exposure at concentrations below the current standard and may suffer long-lasting effects such as stunted lung function in young adulthood (Tager et al., 2005). A national standard of 60 ppb would reduce children's exposures of concern from ozone by 95 to 100 percent. A standard of 60 ppb would provide critical protection for children from the dangers from ozone compared to the current standard, according to EPA's Risk and Exposure Assessment. The strength of that protection draws a stark comparison to the far weaker options of 65 ppb to 70 ppb. By contrast, a standard of 70

ppb would reduce such exposures by only 15 to 35 percent, while a standard of 65 would reduce such exposures by 30 to 65 percent (EPA, Risk and Exposure Analysis, 2014). Older Adults Multiple factors place older adults at greater risk from ozone and other air pollutants, including greater time spent outdoors after age 65, the gradual decline in the functioning of the body's systems that accompany aging and an increase in the responsiveness to ozone (EPA, ISA 2013). Recent studies also added to the existing evidence that older adults face greater risk of premature death from ozone (Medina-Ramón and Schwartz 2008; Zanobetti and Schwartz, 2008a; Cakmak et al 2011). Chronic Disease Individuals with preexisting lung disease face substantial risks. People with asthma, particularly children but also adults, have shown exacerbated respiratory symptoms in multi-city studies (Mortimer et al., 2002, Romieu et al., 1996 and 1997; O'Connor et al., 2008). Studies have tracked increases in hospitalization among adults suffering from chronic obstructive pulmonary disease (Peel et al., 2007; Median-Ramón, et al., 2006). Newer research, in a large, multi-continent study, also shows increased risk of premature death from cardiovascular disease triggered by ozone pollution (Katsouyanni et al., 2009). Comments to Docket ID NO. EPA-HQ-OAR-2008-069 9 Outdoor workers and exercisers Outdoor workers as well as active adults who exercise outdoors (Brauer et al., 1996; Korrick et al., 1998) are particularly vulnerable to ozone exposure due to greater exposure because of time spent outdoors and activity levels. A recent study of lifeguards in Galveston, Texas,

provided evidence of the impact of even short-term exposure to ozone on healthy, active adults. Testing the breathing capacity of these outdoor workers several times a day, researchers found that many lifeguards suffered increased obstruction in their airways when ozone levels were higher (Thaller et al., 2008). Socioeconomic Status Several large studies have identified that individuals who have low socioeconomic status or who live in communities with low socioeconomic status face higher risk of hospital admissions and emergency department visits associated with ozone pollution (Lin et al., 2008; Cakmak et al., 2006b; Burra et al., 2009). As noted earlier, additional studies have identified people who live in communities with high unemployment or other markers of low socioeconomic status as having greater risk of premature death from ozone pollution (Bell and Dominici , 2008; Katsouyanni et al., 2009). Meeting a standard of 60 ppb would provide greater protection to groups already facing substantial challenges. We call on EPA to adopt a standard of 60 ppb The Clean Air Act requires that the EPA set the standard based on the need to protect public health "with an adequate margin of safety." In 2001, the Supreme Court unanimously ruled that protecting health was the only legal basis for the standard. The existing standard fails to protect public health with a margin of safety. EPA must strengthen it. Given the weight of evidence, we urge you to set the eight-hour ozone standard at 60 ppb to protect against known and anticipated adverse health effects and to provide a margin of safety as required by the Clean Air Act.

Sincerely,

American Academy of Pediatrics

American College of Preventive Medicine

American Heart Association

American Lung Association

American Medical Association

American Public Health Association

American Thoracic Society

Asthma and Allergy Foundation of America

Children's Environmental Health Network

Health Care Without Harm

National Association of County and City Health Officials

National Association for Medical Direction of Respiratory Care

Trust for America’s Health.”

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CURRICULUM VITAE
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EDUCATION:

1969	B.A.	Brandeis University
1980	Ph.D.	Brandeis University (Theoretical Physics)
2010	M.D.	University of Basel, Honorus Causa

ACADEMIC APPOINTMENTS:

2005 to present	Professor of Environmental Epidemiology, Harvard School of Public Health Director, Harvard Center for Risk Analysis
1994 -2004	Associate Professor of Environmental Epidemiology, HSPH
1994 -	Associate Professor of Medicine, Harvard Medical School
1991-1993	Lecturer, Department of Environmental Health, Harvard School of Public Health

HOSPITAL APPOINTMENTS:

1994 -	Associate Epidemiologist, Brigham and Women's Hospital

OTHER PROFESSIONAL POSITIONS AND MAJOR VISITING APPOINTMENTS:

1977-1979	Legislative Assistant for Energy and Environment, Congressman Timothy Wirth
1979-1987	Staff Scientist, U.S. Environmental Protection Agency
1987-1988	Visiting Scientist, Department of Biostatistics, Harvard School of Public Health
1989,1994	Visiting Scientist, Department of Social and Preventive Medicine University of Basel, Switzerland
1990	Visiting Scientist, Department of Occupational Safety and Environmental Health University of Wuppertal, Germany
1989-1993	Senior Scientist, U.S. Environmental Protection Agency

HONORS AND DISTINCTIONS:

2008	John Goldsmith Career Achievement Award, International Society for Environmental Epidemiology
2001	Nichols Teaching Award, Harvard School of Public Health
2001	International Union of Environmental Protection Agencies World Congress Award

1999	Twentieth Century Distinguished Service Award, Lukacs Symposium for Statistical Ecology and Environmental Statistics
1991	John D and Catherine T MacArthur Fellowship
1984,1986	U.S. Environmental Protection Agency Silver Medal
1988-89-90-92,14	U.S. Environmental Protection Agency Scientific Achievement Award
1991	Alumni Achievement Award, Brandeis University

MAJOR COMMITTEE ASSIGNMENTS:

National

1985	Preventing Lead Poisoning in Young Children document, Consultant, Centers for Disease Control
1988	Advisory Committee, Boston Soil Lead Study
1989-1992	EPA Environmental Health Review Panel, Environmental Protection Agency
1989-1993	National Academy of Science, Committee on Assessing Lead Exposure in Critical Populations
1990-1993	National Academy of Science, Committee on Environmental Epidemiology
1992	Advisory Committee, Resources for the Future Center for Risk Management, Public Health/ Environmental Health Risk Studies
1992	Environmental Epidemiology Advisory Committee, Pew Memorial Trusts
1992	Ethics Committee, International Society for Environmental Epidemiology
1992	Reviewing Committee, Office of Technology Assessment for Identifying and Controlling Pulmonary Toxicants
1992-6	Technical Advisory Committee, Alliance to End Childhood Lead Poisoning
1992	Technical Advisory Committee, New York State Environmental Externalities Cost Study
1993	Subcommittee on Lead, National Advisory Committee on Environmental Policies and Technology
1993-2002	Research Advisory Committee, National Center for Lead Safe Housing
1994-2002	Center for Disease Control, Advisory Committee on Childhood Lead Poisoning Prevention
1994-2005	Mickey Leland National Urban Air Toxic Research Center, Scientific Advisory Panel
1995-7	Environmental Statistics Subcommittee, National Advisory Committee on Environmental Policy and Technology
1998	Franklin Institute Science Medal Prize Committee
2003-2005	HSPH Disciplinary Committee, Chair
2004-present	Steering Committee, Harvard University Center for the Environment
2005	EPA Science Advisory Board, Ad Hoc All Ages Lead Committee
2005-2008	Councilor, International Society for Environmental Epidemiology
2005-2010	Editorial Board, American Journal of Respiratory and Critical Care Medicine
2005-2008	EPA Lead Clean Air Science Advisory Committee
2014-17	National Research Council. Standing Committee on Use of Emerging Science for Environmental Health Decisions

International

1993-2004	European Economic Community Studies on Air Pollution, Daily Mortality, and Hospital Emergency Visits, Advisor

1993	Advisory Committee, European Economic Community Panel Studies on Air Pollution, Pulmonary Function, and Respiratory Function
2000-2004	Chair, Statistics Advisory Committee, APHEIS Project
2011-2016	Advisory Committee, Southeastern Center for Air Pollution and Epidemiology
2016-present	World Health Organization Global Platform on Air Quality and Health

PROFESSIONAL SOCIETIES:

1987	American Statistical Association
1988	American Thoracic Society
1990	Society for Epidemiologic Research
1991	International Society for Environmental Epidemiology

Editorial Board

2003-4	Epidemiology Editor, International Journal of Biometeorology
2005-2010	Editorial Board, American Journal of Respiratory and Critical Care Medicine

MAJOR RESEARCH INTERESTS:

1. Respiratory Epidemiology
2. Air, Water and Lead Epidemiology
3. Epidemiologic Methods
4. Cost-Benefit Analysis
5. Exposure Assessment

TEACHING EXPERIENCE:

1992-2008	Environmental Epidemiology Course, University of Basel @ 30-40 Graduate Students 35 class hours
1994, 97,99,2009-12,14,18	Advanced Topics in Environmental Epidemiology, University of Basel, @ 20 Graduate Students, 35 class hours
1995	Short Course on Advanced Regression Analysis in Environmental Epidemiology, San Miniato, Italy @ 23 Graduate Students, 35 class hours
1996–present	Professor, ID 271 HSPH, joint course on Advanced Regression Analysis for Departments of Epidemiology, Environmental Health, and Biostatistics, 21 Graduate Students, 35 class hours
2010-present	Professor, EPI 204, Methods for analyzing case control, cohort, and other studies, 100 graduate students, 35 class hours
2007-present	Professor, EH520 Seminar on preparing research proposals
2007-2011,16	Environmental Epidemiology, Cyprus International Institute

1998	European Course on methods for Poisson Time Series, Santorini Greece, @40 Graduate Students, 35 hours
1998	Short Course on Advanced Regression in Environmental Epidemiology, Annual meeting of International Society for Environmental Epidemiology, 45 Graduate Students, 7 hours
1999	European Course on methods of Meta-analysis. Santorini Greece, 40 students, 24 course hours
2006	Environmental Epidemiology, National Institute of Public Heath, Mexico 40 students 30 hours
2012	Topics in Environmental Epidemiology, Summer Institute, Tel Aviv University
2015	Causal Inference in Epidemiology, Sapiensa University, Rome Italy
2016,17	Environmental Epidemiology, Public Health Foundation of India, 48 hours
2018	Causal Modeling in Environmental Epidemiology, Public Health Foundation of India, 35 hours

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Original Reports: (H-index=137)

1. Pirkle J, **Schwartz J**, Landis R, Harlin W. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. Am J Epid 1985;121:246-258.

2. **Schwartz J**, Angle C, Pitcher H. Relationship between childhood blood lead levels and stature. Pediatrics 1986;77:281-288.

3. Marcus A, **Schwartz J**. Dose-response curves for erythrocyte protoporphyrin vs blood lead: Effect of iron status. Environ Res 1987;44:221-227.

4. **Schwartz J**, Otto D. Blood lead levels, hearing thresholds, and neurological development in NHANES II children. Arch Environ Health 1987;42:153-162.

5. **Schwartz J**, Landrigan PJ, Silbergeld E, Feldman R, Baker E, Von Lindern I. Does lead-induced peripheral neuropathy exhibit a threshold? J Pediatrics 1988;112:12-17.

6. Silbergeld E, **Schwartz J**, Mahaffey K. Lead and osteoporosis: Mobilization of lead from bone in postmenopausal women. Environ Res 1988;47:79-94.

7. **Schwartz J**, Haselblad V, Pitcher H. Air pollution and morbidity: A further analysis of the Los Angeles student nurses data. JAPCA 1988;38:158-162.

8. Davis D, **Schwartz J**. Trends in cancer mortality in U.S. white males and females, 1968-1983. Lancet 1988;633-636.

9. **Schwartz J**. The relationship between blood lead and blood pressure in the NHANES II survey. Environ Health Persp 1988;78:15-22.

10. **Schwartz J**, Katz S, Fegley R, Tockman M. Sex and race differences in the development of lung function. Am Rev Respir Dis 1988;138:1415-1421.

11. **Schwartz J**, Katz S, Fegley R, Tockman M. Analysis of spirometric data from a national sample of healthy 6-24 year olds. Am Rev Respir Dis 1988;138:1405-1414.

12. **Schwartz J**. Lung function and chronic exposure to air pollution: A cross-sectional analysis of NHANES II. Environ Res 1989;50:309-321.

13. **Schwartz J**, Pitcher H. The relationship between gasoline lead and blood lead in the United States. J Off Stat 1989;5:421-431.

14. **Schwartz J**, Landrigan PJ, Baker EL, Orenstein WA, Von Lindern IH. Lead induced anemia: Dose-response relationships and evidence for a threshold. Am J Pub Health 1990;80:165-168.

15. Davis DL, Hoel D, Percy C, Ahlbom A, **Schwartz J**. Is brain cancer mortality increasing in industrial countries? Ann New York Acad Sci 1990;609:191-204.

16. **Schwartz J**. Multinational trends in cancer mortality rates: Methodological issues and results. Ann New York Acad Sci 1990;609:136-145.

17. **Schwartz J**. Multinational trends in multiple myeloma. Ann New York Acad Sci 1990;609:215-224.

18. Crocetti AF, Mushak P, **Schwartz J**. Determination of numbers of lead-exposed women of childbearing age and pregnant women. Environ Health Perspect 1990;89:121-124.

19. Crocetti AF, Mushak P, **Schwartz J**. Determination of numbers of lead-exposed U.S. children by areas of the United States. Environ Health Perspect 1990;89:109-120.

20. **Schwartz J**, Marcus A. Mortality and air pollution in London: A time series analysis. Am J Epid 1990;131:185-194.

21. **Schwartz J**, Zeger S. Passive smoking, air pollution, and acute respiratory symptoms in a diary study of student nurses. Am Rev Respir Dis 1990;141:62-67.

22. **Schwartz J**, Gold D, Dockery DW, Weiss ST, Speizer FE. Predictors of asthma and persistent wheeze in a national sample of U.S. children: Association with social class, perinatal events and race. Am Rev Respir Dis 1990;142:555-562.

23. **Schwartz J**, Weiss ST. Dietary factors and their relationship to respiratory symptoms. NHANES II. Am J Epid 1990;132:67-77.

24. **Schwartz J**. Lead, blood pressure, and cardiovascular disease in men and women. Environ Health Perspect 1991;91:71-77.

25. **Schwartz J**, Dockery DW, Wypij D, Ware J, Zeger S, Spengler J, Speizer FE, Ferris BG Jr. Daily diaries of respiratory symptoms and air pollution: Methodological issues and results. Environ Health Perspect 1991;90:181-188.

26. **Schwartz J**, Levin R. The risk of lead toxicity in homes with lead paint hazard. Environ Res 1991;54:1-7.

27. Chestnut LG, **Schwartz J**. Savitz DA, Burchfiel CM. Pulmonary function and ambient particulate matter: Epidemiological evidence from NHANES I. Arch Environ Health 1991;46:135-144.

28. **Schwartz J**, Otto D. Lead and minor hearing impairment. Arch Environ Health 1991;46:300-305.

29. **Schwartz J,** Spix C, Wichmann HE, Malin E. Air pollution and acute respiratory illness in five German communities. Environ Res 1991;56:1-14.

30. **Schwartz J**. Particulate air pollution and daily mortality in Detroit. Environ Res 1991;56:204-213.

31. **Schwartz J**, Weiss ST. Host and environmental factors influencing the peripheral blood leukocyte count. Am J Epid 1991;134:1402-1409.

32. **Schwartz J**, Dockery DW. Particulate air pollution and daily mortality in Steubenville, Ohio. Am J Epid 1992;135:12-20.

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45. **Schwartz J**. Air pollution and daily mortality in Birmingham, Alabama. Am J Epid 1993;137:1136-1147.

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49. **Schwartz J**. Air pollution and daily mortality: A review and meta-analysis. Environ Res 1994;64:36-52.

50. **Schwartz J**. Low level lead exposure and children's IQ: A meta-analysis and search for a threshold. Environ Res 1994; 65:42-55.

51. **Schwartz J**, Dockery DW, Neas LM, Wypij D, Ware JH, Spengler JD, Koutrakis P, Speizer FE, Ferris BG Jr. Acute effects of summer air pollution on respiratory symptom reporting in children. Am J Respir Crit Care Med 1994; 150:1234-1242.

52. **Schwartz J**. Societal benefits of reducing lead exposure. Environ Res 1994; 66:105-124.

53. **Schwartz J**. $PM_{10,}$ ozone, and hospital admissions for the elderly in Minneapolis-St. Paul, Minnesota Arch Environ Health 1994; 49:366-374.

54. **Schwartz J**. Air pollution and hospital admissions for the elderly in Birmingham, Alabama. Am J Epid 1994; 139:589-598.

55. Leuenberger P, **Schwartz J**, Ackermann-Liebrich U and the SPALDIA Team. Passive smoking exposure and chronic respiratory symptoms in adults. Am J Respir Crit Care Med 1994; 150:1222-1228.

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Pub Health 1995;85(11):1343-1345.

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78. Pope CA III, **Schwartz J**. Time series for the analysis of pulmonary health data. Am J Resp Crit Care Med 1996;154:S229-S233.

79. **Schwartz J**, Spix C, Touloumi G, Bacharova L, Barumamdzadeh T, le Tertre A, Piekarksi T, Ponce de Leon A Ponka A, Rossi G, Saez M, Schouten JP. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. J Epid Comm Health 1996;50(1):S3-S11.

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81. **Schwartz J**. Air pollution and hospital admissions for cardiovascular disease in Tucson. Epidemiol 1997; 8:371-377.

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85. **Schwartz J**, Levin R, Hodge K. Drinking water turbidity and pediatric hospital use for gastrointestinal illness in Philadelphia Epid 1997;8:615-620.

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Ahmed Gomaa	“Pre and Postnatal lead exposure and children’s cognitive function”
Sahar Elmarsafawy	“Interaction of lead and calcium in predicting hypertension”
Alfesio Braga	“The time course of weather related deaths”
Yon-Chul Hong	“Effects of Air Pollutants on Acute Stroke Mortality”
Babette Brumback	“Transitional Regression Models with Application to Environmental Time Series”
Ariana Zeka	“Effects of Air pollution on Pregnancy Outcomes”
Andrea Baccarelli	“Cardiovascular Effects of Air Pollution”
Mercedes Medina-Ramon	“Effects of traffic pollution on survival of heart failure patients”
Alexandros Gryparis	“Spatio-Temporal modeling of traffic related air pollution and birth outcomes”
Nicos Middleton	“Traffic related pollution and mortality”
Stephanie von Klot	“GIS based Exposure to Heat, Mortality, and Myocardial Infarctions”
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John McCracken	“Cardiovascular Effects of Wood Smoke Exposure in Guatemala”
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Jaana Halonen	“ Physiologic effects of Temperature”
Itai Kloog	“Spatio-temporal particle models and health”
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